TO EVALUATE THE ROLE OF ABSOLUTE EOSINOPHIL COUNT AS A PROGNOSTIC MARKER IN PATIENTS OF PERFORATION PERITONITIS

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I hereby declare that the thesis entitled **"TO EVALUATE THE ROLE OF ABSOLUTE EOSINOPHIL COUNT AS A PROGNOSTIC MARKER IN PATIENTS OF PERFORATION PERITONITIS"** submitted towards partial fulfillment of the requirements for the award of **MS GENERAL SURGERY** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. Ravinder Pal Singh,** Professor, Department ofSurgery, **Dr. Atul Mishra**, Professor, Department of Surgery.**Dr. Amandeep S. Nar**, Associate Professor, Department of Surgery. No part of this thesis has formed the basis for the award of any degree previously.

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Dedicated

to

my family

&

Teachers

LIST OF ABBREVIATIONS

AEC	Absolute eosinophilic count
CAPD	Continous Ambulatory Peritoneal Dialysis
AIDS	Acquired immunodeficiency Syndrome
PAN	Polyarteritisnodosa
SLE	Systemic Lupus Erythematosus
SBP	Spontaneous Bacterial Peritonitis
NSAIDS	Non steroidal anti-inflammatory drugs
CXR	Chest X Ray
ATT	Anti Tubercular Treatment
ATP	Adenosine Triphosphate
APACHE	Acute Physiology and Chronic health evaluation
COX	Cyclooxygenase
DPL	Diagnostic peritoneal lavage
ABG	Arterial Blood Gas
MODS	Multiple Organ Dysfuction Syndrome
TNF	Tumour Necrosis Factor
FAST	Focused Assessment in Sonography for Trauma
ICAM	Intercellular Adhesion Molecule
VCAM	Vascular Cell Adhesion Molecule

ABSTRACT

Introduction: Peritonitis is the commonest cause of sepsis in patients admitted in surgical wards in developing countries. Cost constraints prevent routine use of expensive tests in developing countries. Eosinopenia has been, recently, found to have strong association with inflammatory-syndrome-associated bacterial infectious diseases. This prompted us to prospectively assess the diagnostic value of eosinopenia as a prognostic marker in perforation peritonitis patients.

Aim of Study: To identify high risk patients and correlate morbidity mortality with eosinophil count.

Materials & methods: This observational study was conducted in Department of Surgery, Dayanand Medical College & Hospital, Ludhiana involving patients who came to casualty or OPD with perforation peritonitis in the period from 01/02/2021 to 31/07/22. AEC values were recorded at the time of admission 24 hours, 72 hours and at the time of discharge. The correlation between eosinophilic count and the final outcome that is discharge or death was studied for any correlation.

Results: 101 patients were allocated into groups based on the outcome as mortality or survival. 85 patients were in the survival group and 16 patients were in the mortality group. There was statistically significant difference in AEC between the two with p value < 0.05 which was statistically significant.

Conclusion: AEC is a reliable marker of survival and it allows timely identification of high risk patients. It can be used as a marker for risk stratification in perforation peritonitis patients. AEC has the necessary sensitivity and specificity in addition to easy methodology and cost effectiveness as seen with other markers of sepsis.

Keywords: Absolute eosinophil count, Perforation peritonitis, Mortality, Morbidity.

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Dr. Aseem Gumber

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INTRODUCTION

Perforation peritonitis is a major surgical emergency that is prevalent in India.[1] Perforation is defined as an abnormal opening in a hollow organ or a hole or a break in the containing wall or membrane of an organ or structure of the body It is derived from the Latin word perforatus, meaning “to bore through“ and is characterized by inflammation of the peritoneum, tissue that lines the abdomen.

The spectrum of etiology of perforation is different between developing and developed countries.[2-3] In India, perforation peritonitis is commonly seen in younger age groups.[4], and the proximal part of the gastrointestinal tract is the most commonly affected site, whereas in Western countries, the distal part is most commonly affected. The most common causes of perforation peritonitis in India are peptic ulcer, typhoid, and appendicular and tubercular perforations.[4] On the other hand, in Western countries, other factors like diverticulitis and colorectal cancer contribute more to the incidence of perforation peritonitis.

The signs and symptoms of almost all cases of perforation peritonitis are typical and clinical diagnosis of peritonitis can be made in all patients. X ray chest and abdomen, ultrasound abdomen and CT scan are the investigations that can confirm the diagnosis. Peritonitis usually presents as an acute abdomen. Local findings include generalized abdominal tenderness, guarding, rigidity, abdominal distension, decreased bowel sounds. Systemic findings include fever with chills or rigor, restlessness, tachycardia, tachypnea, dehydration, oliguria, disorientation and ultimately shock.

In patient with perforation peritonitis, the prognosis affecting factors are patients’ age, vitals, metabolic acidosis, malnutrition, personal habits of smoking, alcoholism and drug abuse, preoperative status, serum albumin, cause of perforation, site of origin of peritonitis and contamination in

peritoneal cavity. If left untreated, the contamination can rapidly spread into the blood stream causing sepsis and to other organs, resulting in multiple organ failure(MODS) and mortality.

Perforation peritonitis is a complex and challenging condition that requires a multi-disciplinary approach for its management. In addition to prompt diagnosis, appropriate surgical intervention, intensive care support and the use of antimicrobial therapy plays a critical role in reducing the incidence of septicemia and shock, the most important factors contributing to mortality in patients with perforation peritonitis.

Despite the recent advancements in diagnosis, intensive care treatment, surgical techniques, and antimicrobial therapy, the management of perforation peritonitis continues to pose a significant challenge for surgeons in India. The incidence of peritonitis is the highest cause of sepsis in developing countries like ours, and the mortality rates continue to remain high, reaching up to 40%. This high mortality rate is attributed to a combination of various factors such as late presentation of patients to clinics, limited availability of sensitive and specific blood biomarkers, and septicemia. In a developing country like ours majority of the cases present late to the hospitals , More so with well established generalized peritonitis with purulent or fecal contamination and septicemia of varying degree, making the surgical management of these cases highly demanding and even more complex and increasing the overall morbidity and mortality of the disease in 30-50% cases.[5]

Another significant challenge for accurate and timely diagnosis of perforation peritonitis is the limited availability of sensitive and specific blood biomarkers. This creates a pressing need for a tool that would predict the morbidity and mortality in patients with perforation peritonitis.

A rapid and persistent decrease in the number of circulating eosinophils, known as eosinopenia, is a distinctive aspect of the physiological response to acute inflammation. Eosinopenia, defined as a count of less than 50 cells per microlitre of blood, may be attributed to the migration of eosinophils into the inflammatory site due to the release of chemotactic factors.

However, there are very few recent studies available in the literature which have highlighted the strong association of eosinopenia with inflammatory-syndrome-associated bacterial infectious diseases and sepsis.[6-9] The use of eosinopenia as a marker for perforation peritonitis and sepsis in India has the potential to be a cost-effective alternative to the procalcitonin assay, which is not widely used in developing countries due to cost constraints. This finding prompted us to prospectively assess the diagnostic value of eosinopenia as a mortality and prognostic marker in patients with perforation peritonitis presenting in our institution.

AIM AND OBJECTIVES

To timely identify high risk patients so as to correlate morbidity and mortality with absolute eosinophilic count

REVIEW OF LITERATURE

History

Ebers papyrus [Egypt] 1500 BC contains the first description of peritoneum. Douglas in 1730 was the first to give the detailed description of peritoneum, thereafter various personalities gave their contribution in its structure, function and pathological conditions.

- Mikulicz (1889) first proposed the basic principles of management of peritonitis involving early operation, source control and lavage. Kirschner et al reported that mortality decreased from 80-100% to about 60% with the introduction of operative procedures.[10] In 1980 ,Fry et al showed that mortality based on number of failed organs. Mortality was 3% when no organ failure, which increased to 30% with one major organ failure and to 100% with four major organ failures.[11]

PERITONITIS

"Intra abdominal infection may be defined as clinical peritonitis with operative and microbiological proof of infection" [12,13] The name includes a group of pathologies namely primary, secondary, tertiary peritonitis and intra abdominal abscess.(Table 1) In spite of research and developments in surgical management, antibiotics, and intensive care treatment, death rate of 5-40% are reported.[12-20]

TABLE- I Type of pathology in peritonitis

I. Primary Peritonitis A. Spontaneous peritonitis in Children B. Spontaneous peritonitis in Adults	II. Tertiary Peritonitis A. Peritonitis without pathogens B. Peritonitis with fungi

C. Spontaneous peritonitis in CAPD D. Tubercular peritonitis	
II. Secondary Peritonitis A. Acute Perforation peritonitis 1.GI tract perforation 2.Intestinal ischemia 3.Pelvic peritonitis 4.Others B. Post operative peritonitis 1.Anastomotic leak 2.Stump insufficiency 3.Other iatrogenic leaks C. Post traumatic peritonitis 1. After blunt abdominal trauma 2. After penetrating abdominal trauma	IV. Intarabdominal Abscess A. Associated with Primary Peritonitis B. Associated with Secondary Peritonitis C. Associated with Tertiary Peritonitis

PRIMARY OR NONSURGICAL BACTERIAL PERITONITIS

Occurring in the absence of gastrointestinal perforation is caused mainly by haematogenous spread but occasionally by transluminal or direct bacterial invasion of the peritoneal cavity. Impairment of the hepatic reticulo endothelial system and compromised peripheral destruction of bacteria by neutrophils promotes bacteremia, which readily infects ascitic fluid that has reduced bacterium-killing capacity.

PRIMARY PERTONITIS- ETIOLOGY

1. Spontaneous bacterial peritonitis,
2. Peritonitis with CAPD,
3. Tuberculous etiology,
4. Peritonitis with AIDS,
5. Chlamydia infection,
6. Gonorrhoea infection,
7. Others- PAN,SLE, Scleroderma, Familial Mediterranean fever, etc

SECONDARY OR SURGICAL BACTERIAL PERITONITIS

The causes of secondary bacterial peritonitis include diseases of the bowel or injury to the bowel, leading onto discontinuity of the GIT and spillage of these contents into peritoneal cavity.[21] This is further divided into acute suppurative peritonitis, granulomatous peritonitis, and chemical peritonitis.[22]

Systemic sepsis due to peritonitis occurs in varying degrees depending on the virulence of the pathogens, the bacterial load, and the duration of bacterial proliferation and synergistic interaction. Surgical peritonitis is diagnosed when ascitic fluid culture is positive for multiple organism, PMN count> 250 cells/mm3 & intra-abdominal surgically treatable primary source of infection. [23]

In most of the cases the peritoneal contamination is caused by mixed flora both aerobic and anaerobic. Anatomical, pathological, and surgical factors may favour localization of peritonitis .However , in majority of the cases peritonitis becomes diffuse when it occurs in patients with sudden anatomical disruption, extremes of age, immunodeficiency , perforation proximal to obstruction, stimulation of peristalsis and following trauma. The clinical presentation of the patients also depends upon the site of

perforation. Patients of duodenal perforation present with a short history of pain epigastrium or upper abdomen along with generalized tenderness and guarding. Patients of diverticulitis are generally of older age and past history of constipation is present along with signs of peritonitis. Appendicular perforations have a characteristic pain starting in periumbilical area or right iliac fossa along with vomiting and fever. There are also conspicuous signs present like guarding and rebound tenderness in right iliac fossa. Ileal perforations are usually preceded by a history of some medical disease followed by sudden onset of lower abdomen pain, vomiting, abdominal guarding and distention later on. In patients of trauma generalized peritoneal signs start developing after 2-3 hours of injury.

HISTORY & PHYSICAL EXAMINATION

Abdominal pain-character, site, radiation, change over time, aggravating & relieving factors.[24]

- Nausea, Vomiting[ileus],
- Abdominal movements absent [pain],
- High fever >100^{o}F [host defence] ,
- Hypovolemia signs- tachycardia, dry mucus membrane, and hypotension,
- Absent liver dullness,
- Board like abdominal rigidity,
- Involuntary guarding,
- Rebound tenderness,
- Absent bowel sounds,
- Rectal and pelvic examination

MANAGEMENT OF PERITONITIS

“Early diagnosis with prompt surgical intervention and aggressive preoperative and postoperative management is essential to reduce the morbidity and mortality from multiorgan system failure resulting from untreated peritonitis” [11,25,26,27]

As soon as diagnosis is made, immediate fluid resuscitation and antibiotic therapy started, patient should be prepared for laparotomy or laparoscopy. Adequate fluid resuscitation is monitored by blood pressure, heart rate, urine output, central venous pressure and pulmonary capillary wedge pressure measurement.

Antibiotics should cover gram-ve aerobes and anaerobes. Multiple drug regimens either single or in combination are recommended, with comparable efficacy, by the Surgical Infection Society for treating intra-abdominal infections.[28] Single broad spectrum antibiotics like beta lactams, fluoroquinolones, third or fourth generation cephalosporins are adequate, routine use of nephrotoxic aminoglycoside not indicated based on recent cochrane review by Wong PF et al.[29] The use of antifungal drugs against candida infection is indicated only in septic shock and immunocompromised patients.

Nosocomial infection due to multiresistant Pseudomonas, Enterobacter, Enterococcus, Staphylococcus and Candida species can occur due to long hospital stay. When patient develop MODS after surgery, thorough search for missed source or abscess, by repeating CT, followed by abscess drainage by percutaneous or operative method, fluid culture and appropriate antibiotics are to be done.[30]

SURGERY

Antibiotics cannot cure the leaking gut or sterilize the abscess, so surgery is the treatment of choice, should be initiated when patient became hemodynamically stable. The aim of surgery is elimination of source, peritoneal lavage and prevent of reinfection.

Surgical treatment forms the mainstay of treatment.[31] Once the general condition of the patient is stabilised, laparotomy must be done to wash the peritoneal cavity copiously and also for closure of the perforation. If the infective focus is not cleared, debridement is partial, or definite abdominal closure is questionable, laparotomy in multiple stages may be needed for debridement, removal of abdominal packs, and repeated washing irrigation till the focus of infection is brought under control and abdominal closure is achieved. Placement of large abdominal zipper is of great help for repeated opening of the abdomen .

POST-OPERATIVE CARE-

Fluid and nutritional support should be initiated. Antibiotics should be continued & changed based on culture report. It is important to maintain a positive nitrogen balance preferably by enteral feeding using feeding jejunostomy.

PROGNOSIS

Mortality rate for secondary peritonitis is 14%, low for appendicitis and perforated duodenal ulcer[10%] and high for tertiary peritonitis[50%].

HEMATOLOGICAL MARKERS IN PERITONITIS

“Eosinophils were first detected in peripheral blood in the year 1865 by Max Schultze.[32] A specific stain for staining the granules was described by Ehrlich P. in 1879”.[33]

“By 1888 observers were studying differential counts of leukocytes. During this period Mayet described his eosin-glycerine method. It was in 1910 that Dunger[34] established the eosinophil counting method using the counting chamber”.

The cellular elements of the blood are produced from common multipotential hematopoietic cells. These cells undergo mitotic division and maturation to form cellular elements of the blood, namely the erythrocytes, leukocytes and thrombocytes.

On the basis of function leukocytes are divided into the granulocytic, monocytic and lymphoid series. The granulocytic leukocytes are further subdivided on the basis of morphology into neutrophils, eosinophils and basophils.

MATURE FORMS OF GRANULOCYTES:

The granules are normally produced by rough endoplasmic reticulum and transported to golgi apparatus for packing. The band forms have a typical elongated nucleus. The segmented neutrophils has a characteristic multilobulated nucleus and contain antibacterial substances, including hydrolases, lysozyme and myeloperoxidase.

The eosinophils are bilobed and differ from neutrophils in that they lack lysozyme. These granules are of two types:

- Small round granules, which have been identified as not containing crystalloids. These granules exist in small quantities in mature eosinophils and are rich in acid phosphatase.
- Large crystalline granules, which are more numerous. These crystalline granules are elliptical, larger than granules of the neutrophil, and had an amorphous matrix surrounding an internal crystalline structure. These crystals are thought to represent the enzyme peroxidase and the matrix contains acid phosphatase.

Basophils contain heparin and histamine.[35]

NORMAL VALUE OF GRANULOCYTE:

1. Neutrophils- 1.50- 7.50* 109 /L [40-75%]

2. Eosinophils – 0.05 – 0.06 * 109 /L[1-7%]

3. Basophils- 0.02 -0.05* 109 /L[0-2%]

Pathogenesis in infection

Infection with the microbe activates the defence mechanism where the cells of the immune system get access to the site of infection . This is followed by activation of the complement component C3b, which forms a complex with the microbe. The neutrophil chemo attractant and activator C5a together with C3a and C4a causes degranulation of mast cells releasing histamine . Histamine release causes contraction of smooth muscles and rapid increase in vascularity.

The pro inflammatory mediators are released by the immune cells and also by the microbe and the damaged cells. These mediators upregulate the expression of intercellular adhesion molecules on vascular endothelium, causing adhesion of the immune cells to the vascular endothelium[rolling]. The L- selectin cell surface adhesion molecule on the

neutrophils recognizes sialy llewis residues on the vascular endothelium. This activates the neutrophil, followed by rapid shedding of the L-selectin from its surface and expresses another cell surface adhesion molecule [e.g., integrins]. The inflammatory mediators [e.g., bacterial lipopolysaccha ride, and the cytokine, interlukin-1 and tumour necrosis factor α] stimulates those integrins to bind E-selectin on the vascular endothelium. Complement system and chemokines contribute to the recruitment of inflammatory cells.

Activated neutrophils transmigrate through the vessel wall to the site of infection where they phagocytose the C3b- coated microbes. Mutations in the genes coding various cell to cell adhesion molecules were found in leukocyte adhesion deficiencies, and this may be the reason for severe life threatening infections.

Sepsis

Neutrophils are double edged weapons. Although neutrophils are essential for the phagocytosis of pathogens , excessive release of proteases and oxidants by these cells are responsible for the injury to various organs.

Early biochemical event in sepsis is the release of cytokines like TNF, Interleukins. Of these TNF,IL-1,IL-8 are considered pro inflammatory, whereas IL-6,IL-10 are anti-inflammatory. Microbial toxin triggers the production of TNF and IL-1 which in turn promotes endothelial cell leukocyte adhesion followed by the release of proteases and arachidonate metabolites and activation of clotting.

Eosinophils

Eosinophils are considered as homeostatic regulators of inflammation. They leave the circulating blood when adrenal cortical hormone increases.

Functionally this means that the eosinophils attempts to suppress inflammatory tissue reactions to prevent the excessive spread of inflammation.

Eosinophils proliferate in response to antigenic stimulation and contain substances that inactivate factors released by mast cells and basophils. The primary function of eosinophils appears to be their reactions with products from mast cells, lymphocytes, and other soluble substances in the blood such as the clotting factors, complement components and hormones.

Although eosinophils are ineffective in protecting the body against invading foreign particles, they do play a role in the defense mechanisms against infections. Eosinophils have the ability to interact with the larval stages of some helminthic parasites and damage them by means of oxidative mechanisms. Certain proteins released from eosinophilic granules are known to damage antibody coated Schistosoma parasites.

METHOD

The Dunger's solution is used for staining of eosinophils which contains acetone, Eosin and distilled water.

Acetone 10 ml and aqueous eosin 0.1gm are the reagents that are mixed in equal quantity. The reconstituted stain should be used within 4 hours and there should be no precipitation . When viewed under microscope the eosinophilic granules stain red, while other leucocytes nuclei stained blue-green.

The determination of complete blood count is by manual counting. Although this is cost effective, this method increases the work load and is time consuming. The manual counting is now replaced by automation decreasing the turn around time to 80-120 hematology samples/hr, and 18-24 tests per sample are done with just 100 µ1 of blood.

EOSINOPENIA AS A MARKER OF SEPSIS

The clinicians need sufficient tests to promptly diagnose sepsis, as early diagnosis and treatment will significantly reduce the mortality and morbidity[36] An early diagnosis of septicemia without microbial culture would ensure early administration of antibiotic therapy,this in turn reduces the patient mortality. Unfortunately, till now there is no marker of infection that satisfies the need as sensitive and specific marker.[37,38] An ideal marker of infection should have increased sensitivity and specificity, easy methodology, short run time, cost effective, and should be in concordance with the severity and prognosis of infection.

It is a well known fact that in acute infection/ inflammation there is rapid decrease in the number of circulating eosinophils.[39] This marked reduction in the number of circulating eosinophil leucocytes in acute infection was first described by Zappert in 1893[40], and widely used in the last century as a useful diagnostic sign.[41]This was followed later by the information that eosinopenia is a part of the normal response to stress[42], it was said that eosinopenia in acute infection is a secondary response to stress induced by the infection.[43]." The value of this old marker of acute infection was tested by Gil and colleagues.[44]

Gold standard test for the identification of sepsis has not been identified so far; but Procalcitonin is found to play a promising role in septicemia and it is found to complement clinical findings and lab test results in cases of septicemia.[45-47] The disadvantage of procalcitonin assay is its cost, and because of the high cost this test did not gain popularity in developing countries and it cannot done in our hospitals. Many studies show that the good old marker of sepsis, decreased absolute eosinophil count being cheaper performs equally well as procalcitonin in the diagnosis of sepsis.

The study by Gil and colleagues in a department of internal medicine showed that an inflammatory syndrome associated with an eosinophil

count <40 cells/mm3 is related to bacterial infectious diseases, eosinopenia was thus used to identify septicemia in this study.[44] In an experimental study, Bass and colleagues produced eosinopenia in rabbits and in humans using chemotactic factors of acute inflammation.[48]"

"In trauma patients, however, DiPiro JT and colleagues found an increased eosinophil count after sepsis.[49] This eosinophil production was enhanced by IL-4 and IL-5, and suggests a T-helper lymphocyte type 2 cytokine activation in response to sepsis after traumatic injury".

Normally 1% to 3% of peripheral blood leucocytes are contributed by eosinophils, and the upper limit of absolute eosinophil count is 350 cells/mm3 blood.[50] The eosinophils level in our body are under regulation. In acute infection ,eosinopenia, occurs as a response to stress, under the influence of corticosteroids and epinephrine. It was also explained that the eosinopenia was the result of recruitment of eosinophils in the circulation to the site of infection. This recruitment occurs as a result of chemokines produced by the immune cells, microbes and damaged tissue in acute infection. The main chemokines being C5a and fibrin fragments whose presence have been found in the circulation in acute inflammation.[48]

A study by SwaminathanSP et al[51] was conducted in Madras Medical College, Chennai involving a total of 104 patients with perforation peritonitis from October 2017 to October 2018 and they were allocated into two groups based on the outcome as mortality or survival. 88 patients were in the survival group and 16 patients were in the mortality group. The Absolute eosinophil count distribution in both these groups were recorded and mean AEC was (cells/cu.mm) 168.64 ±34.84 (107- 242) in survival group and 33.13 ±7.50 (23-45) in mortality group .They concluded that AEC is a reliable marker of survival and it allows timely identification of high-risk patients. It can be used as a marker for risk stratification in perforation peritonitis patients. AEC has the necessary sensitivity and

specificity in addition to easy methodology and cost effectiveness as seen with other markers of sepsis

In another prospective study conducted by Jagdeesh TS et al.[52] involving 94 consecutive patients with perforation peritonitis patients admitted to the surgical wards at a teaching hospital in Central India. Jabalpur prognostic score (JPS),C-reactive protein (mg/dL)levels, and absolute eosinophil counts (cells/mm^3) were measured on admission. When correlated with mortality, univariate analysis showed JPS, CRP, and AEC to be accurate prognostic markers ,while multivariate analysis showed only AEC to be accurate They have used ROC/ AUROC to compare the diagnostic accuracy of JPS,CRP and AEC. They concluded that AEC allows timely identification of patients at high risk for sepsis related mortality in patients with peritonitis.

Abidi K et al.[9] in their study found eosinopenia as an early marker of mortality in critically ill patient . Also he found that eosinopenia is a better marker of blood stream infections in critically ill patients than CRP and procalcitonin.

As eosinophil count is routinely done as a part of complete blood count, it does not entail any extra effort or expenditure. Hence, AEC not only has the sensitivity and specificity but also has the “ease” and “cost effectiveness” not seen with other markers for sepsis.

MATERIAL AND METHODS

Cases with perforation peritonitis in the Department of Surgery, Dayanand Medical College and Hospital, Ludhiana over a period of 1.5 years, was studied and analysed statistically.

Source of Data

The study was conducted involving patients with perforation peritonitis who were admitted in the “Department of Surgery, DMC Hospital, Ludhiana ”within the given time period (Feb 2020 to July 2022) after considering the inclusion and exclusion criteria

Inclusion Criteria

- Patients with secondary bacterial peritonitis due to hollow viscous perforation.

Exclusion Criteria-

1.Spontaneous bacterial peritonitis

2. Malignant perforation.

3.Traumatic perforation

4.Non-resuscitable patients

5.Post surgical leak**.**

Method of collection of data:

All the patients with acute abdomen with clinical signs of peritonitis were investigated for perforation and underwent radiological investigations - chest x-ray and x ray erect abdomen. In case of a few patients , where the x ray abdomen findings were unremarkable , a CT abdomen was done to ascertain the diagnosis of perforation peritonitis. After confirmation of perforation peritonitis, requisition for eosinophilic and absolute eosinophilic count was given along other routine haematological investigations.

At the time of admission,5mL of venous blood was collected in EDTA tube for the determination of Absolute Eosinophil Count (AEC).

The absolute eosinophil count was determined in the automated Beckman coulter DxH 900 by counting the number of eosinophils per 100 white blood cells. It was then multiplied by the total leukocyte count of the patient.

Sample collection duration: At the time of admission
24 hours
72 hours
At the time of discharge

The reports of these along with other vital parameters were recorded everyday.

The correlation between eosinophilic count and the final outcome that is discharge or death was studied for any correlation.

Statistical Analysis : Data was processed using SPSS software. All values were expressed as mean ± Standard deviation, Frequency and percentage. Comparison of absolute eosinophil count between the mortality and survival group was done using Mann whitney test.

OBSERVATION AND RESULTS

A hospital based prospective study was conducted on 101 patients who presented with perforation peritonitis to the casualty or OPD at Dayanand Medical College and Hospital, Ludhiana. Patients were allocated into two groups, mortality and survival based on their clinical outcome.

1. Age Distribution

In our study, majority of the patients were in the younger age group i.e. less than 30 years(23.8%),followed by patients with age between 51-60(22.8%).18 patients were in the age group >70 years(17.8%).14 patients were in the age group 61-70(13.9%),12 patients were in the age group 31-40(11.9%),and least were in the age group 41-50(9.9%).

Table 1: Age distribution

AGE GROUP	No. of cases	Percentage
< 30	24	23.8%
31-40	12	11.9%
41-50	10	9.9%
51-60	23	22.8%
61-70	14	13.9%
> 70	18	17.8%
Total	101	100.0%

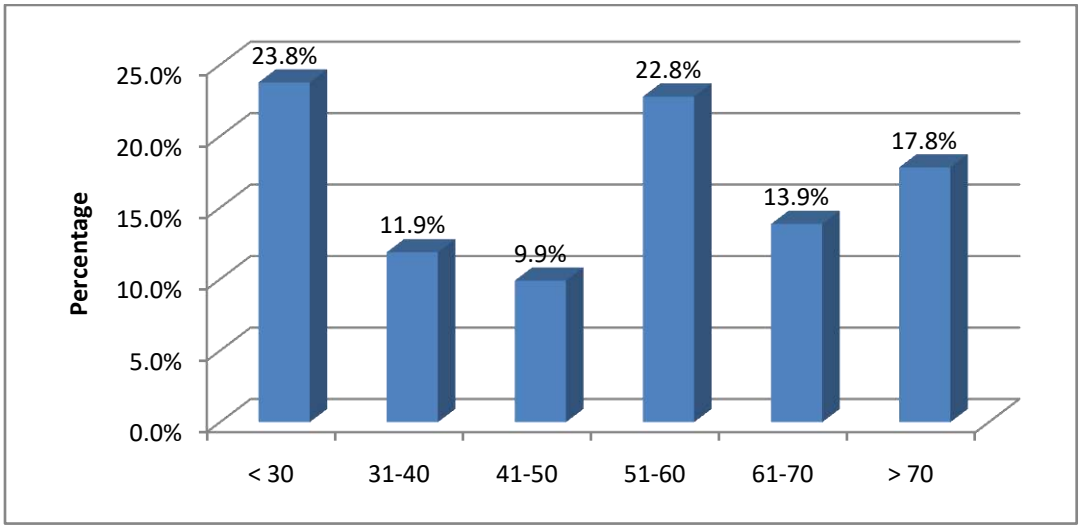

Figure 1 - Age distribution

2. Gender Distribution

In our study there was a male preponderance with number of males being 82(81.2%) and females being 19(18.8%).

Table 2:Showing the sex distribution of the patients presenting with perforation peritonitis.

SEX	No. of cases	Percentage
F	19	18.8%
M	82	81.2%
Total	101	100.0%

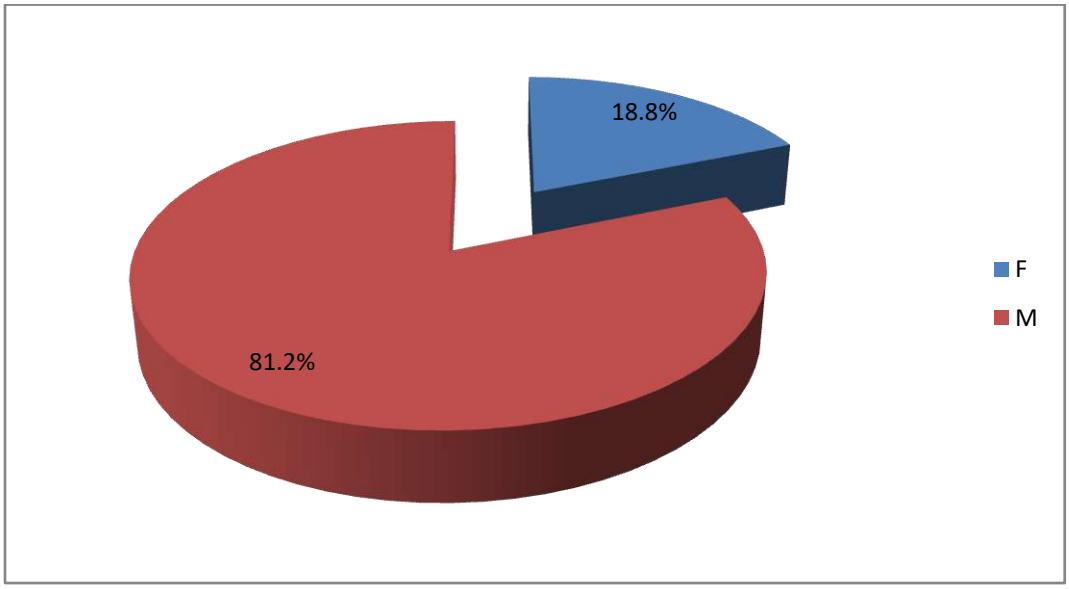

Figure 2 - Showing the sex distribution of the patients presenting with perforation peritonitis.

3. Site of perforation

In our study, the most common site of perforation was peptic (n=33 ,32.7%) ,followed by appendicular (n=28,27.7%),followed by Ileal perforation (n=26,25.7%) and colonic (n=14,13.9%) was least among them.

Table 3 - Shows the type of perforation in patients presenting with perforation peritonitis.

CLINICAL DIAGNOSIS	No. of cases	Percentage
appendicular perforation	28	27.7%
colonic perforation	14	13.9%
ileal perforation	26	25.7%
peptic perforation	33	32.7%
Total	101	100.0%

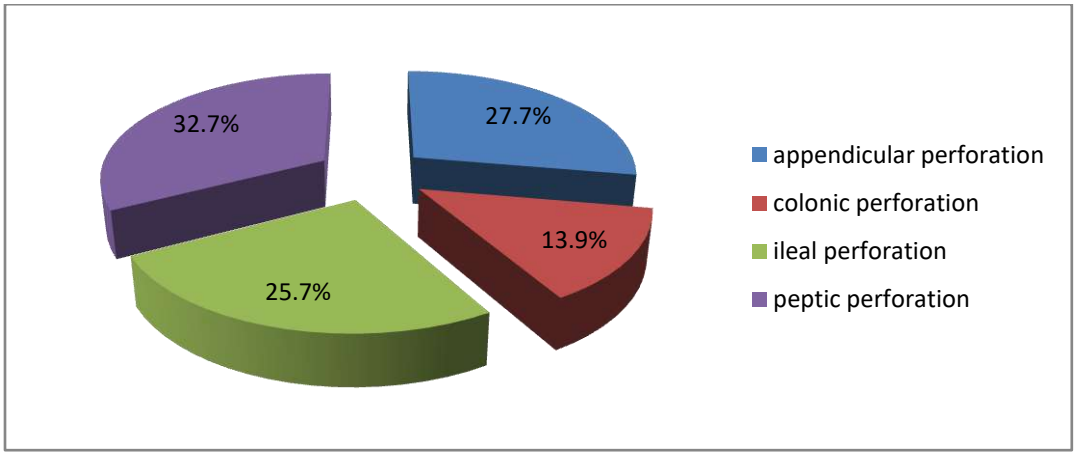

Figure 3 Shows the types of perforation in patients presenting with perforation peritonitis.

4. Overall Mortality

Out of 101 patients in our study who presented with perforation peritonitis mortality was seen in16(15.8%) patients .

Table 4 - Shows the overall mortality in patients with perforation peritonitis.

OVERALL MORTALITY	No. of cases	Percentage
NO	85	84.2%
YES	16	15.8%
Total	101	100.0%

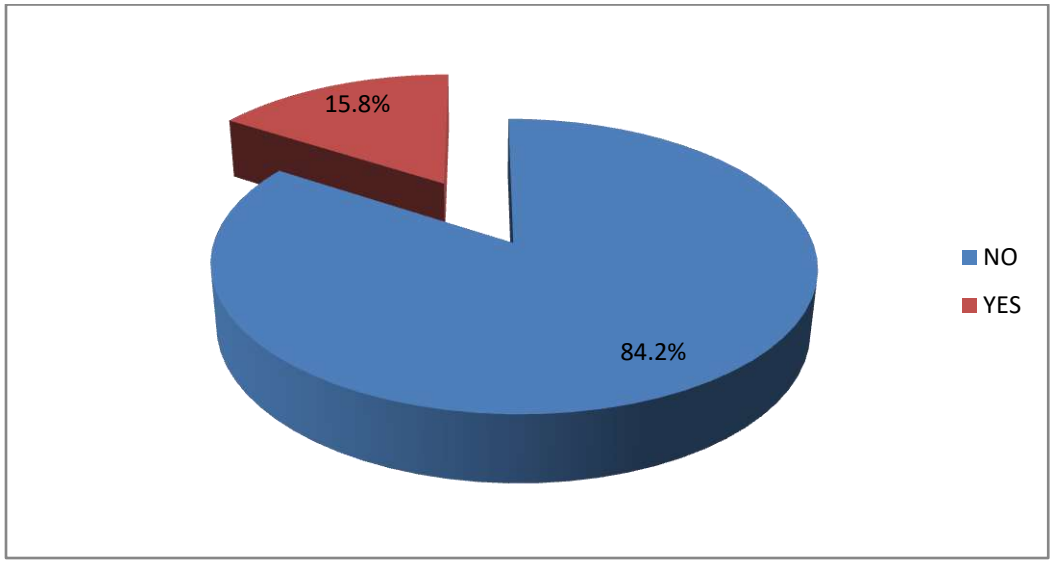

Figure 4 - Shows the overall mortality in patients with perforation peritonitis.

5. Mortality and its relation with age:

In our study, patients in the age group of more than 70 had the maximum number of mortalities i.e.5 out of 16 (31.3%).it was followed by 4 out of 16 (25%)patients in the age group of 31-40, 3 out of 16(18.8%) in the age group of 41-50,followed by 2 out of 16(12.5%) in the patients aged less than 30 and 1 out 16 (6.3%) in the age group of 51-60 and 61-70 each.

Table 5: Shows the relation of mortality with age in patients with of perforation peritonitis.

	MORTALITY	**Survival**		**Non-survival**		**Total**	**Chi-square value**	**p-value**
		No. of cases	**%age**	**No. of cases**	**%age**			
AGE GROUP	< 30	22	25.9%	2	12.5%	24	10.27	0.068
	31-40	8	9.4%	4	25.0%	12		
	41-50	7	8.2%	3	18.8%	10		
	51-60	22	25.9%	1	6.3%	23		
	61-70	13	15.3%	1	6.3%	14		
	> 70	13	15.3%	5	31.3%	18		
Total		85	100.0%	16	100.0%	101		

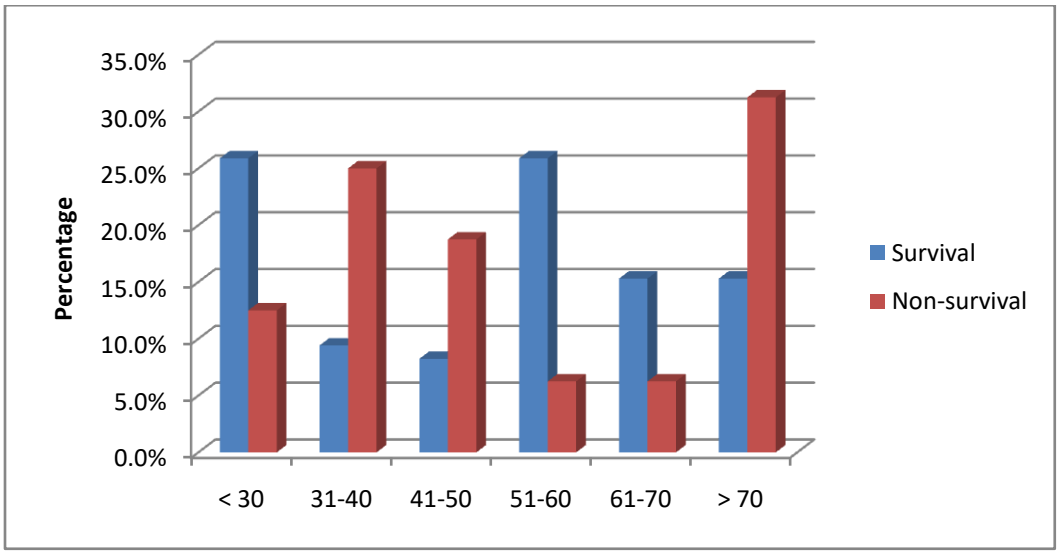

Figure 5: Shows the relation of mortality in patients with of perforation peritonitis.

6. Mortality and its relation with gender:

In our study, mortality was seen in 16 patients, 14(87.5%) of which were males and 2 (12.5%) were females.

Table 6: Shows gender distribution among the patients who had mortality

	MORTALITY	**Survival**		**Non-survival**		**Total**	**Chi-square value**	**p-value**
		No. of cases	**%age**	**No. of cases**	**%age**			
SEX	F	17	20.0%	2	12.5%	19	0.496	0.481
	M	68	80.0%	14	87.5%	82		
Total		85	100.0%	16	100.0%	101		

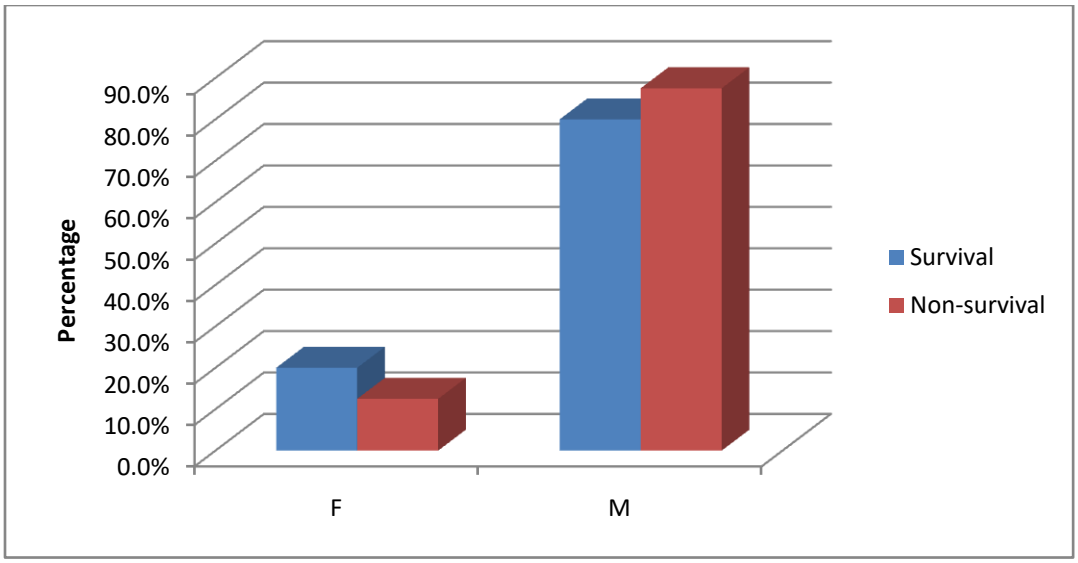

Figure 6: Shows gender distribution among the patients who had mortality

7. **Relation of pre existing co-morbidities with mortality:**

   In our study, out of the 101 patients, 22(21.78%) had pre-existing co-morbidities Asthma(1),Coronary artery disease(2),Diabetes mellitus(3),Typhoid(6),Hypertension(4) and Hypertension with Diabetes(6).Out of which 1(6.3%) with coronary artery disease had mortality,2(12.5%) with diabetic mellitus had mortality,2 (12.5%) with typhoid had mortality,1(6.3%) with hypertension had mortality and 3(18.8%) with both hypertension and diabetes had mortality.

Table 7: Shows relation of pre existing co-morbidities with mortality

	MORTALITY	Survival		Non-survival		Total	Chi-square value	p-value
		Survival	Survival	Non-survival	Non-survival			
MEDICAL	asthma	1	1.2%	0	0.0%	1	17.519	0.008
	CAD	1	1.2%	1	6.3%	2		
	DM	1	1.2%	2	12.5%	3		
	H/O TYPHOID	4	4.7%	2	12.5%	6		
	hypertensive	3	3.5%	1	6.3%	4		
	Hypertensive + DM	3	3.5%	3	18.8%	6		
	NIL	72	84.7%	7	43.8%	79		
Total		85	100.0%	16	100.0%	101		

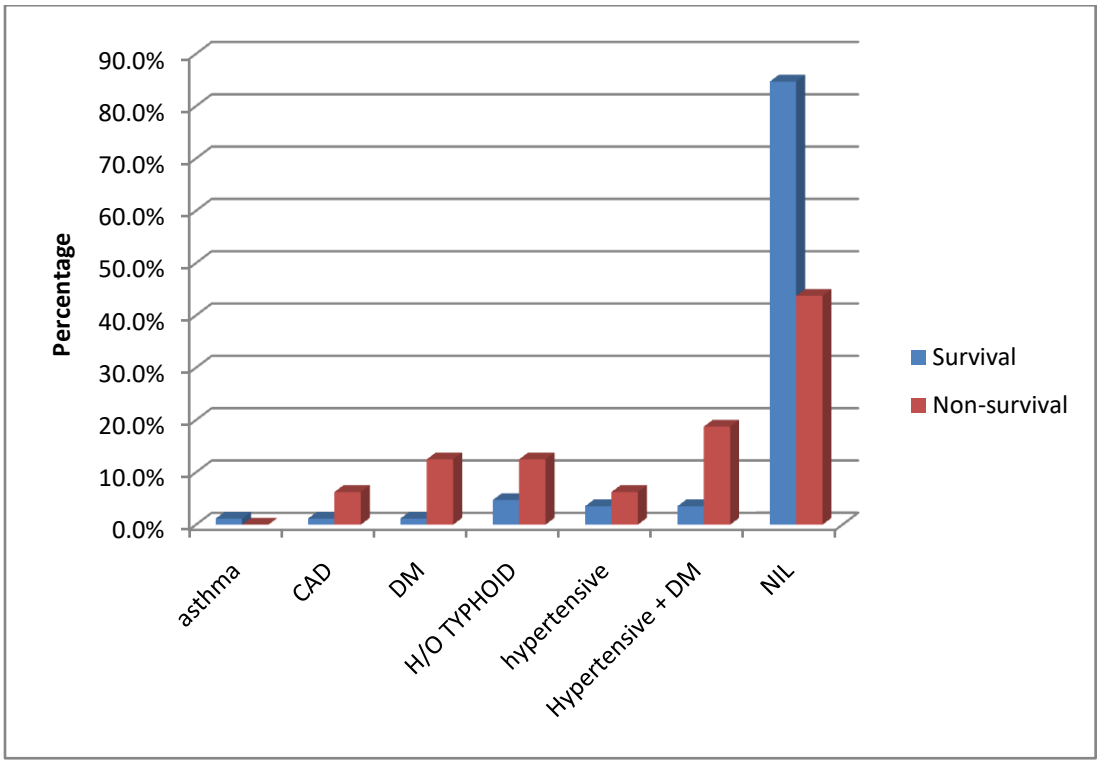

Figure 7:Shows relation of pre existing co-morbidities with mortality

8. Mortality and its correlation with site of perforation:

In our study, majority of the mortality cases were seen in patients presenting with peptic perforation 6(37.5%),followed by ileal perforation 5(31.3%),appendicular perforation 4(25%) and 1(6.3%) with colonic perforation resulted in mortality.

Table 8: Shows the Mortality pattern based on site of perforation

		OVERALL MORTALITY				Total	Chi-square value	p-value
		NO		YES				
CLINICAL DIAGNOSIS	Appendicular Perforation	24	28.2%	4	25.0%	28	1.919	0.751
	colonic perforation	13	15.3%	1	6.3%	14		
	ileal perforation	21	24.7%	5	31.3%	26		
	peptic perforation	27	31.8%	6	37.5%	33		
Total		85	100.0%	16	100.0%	101		

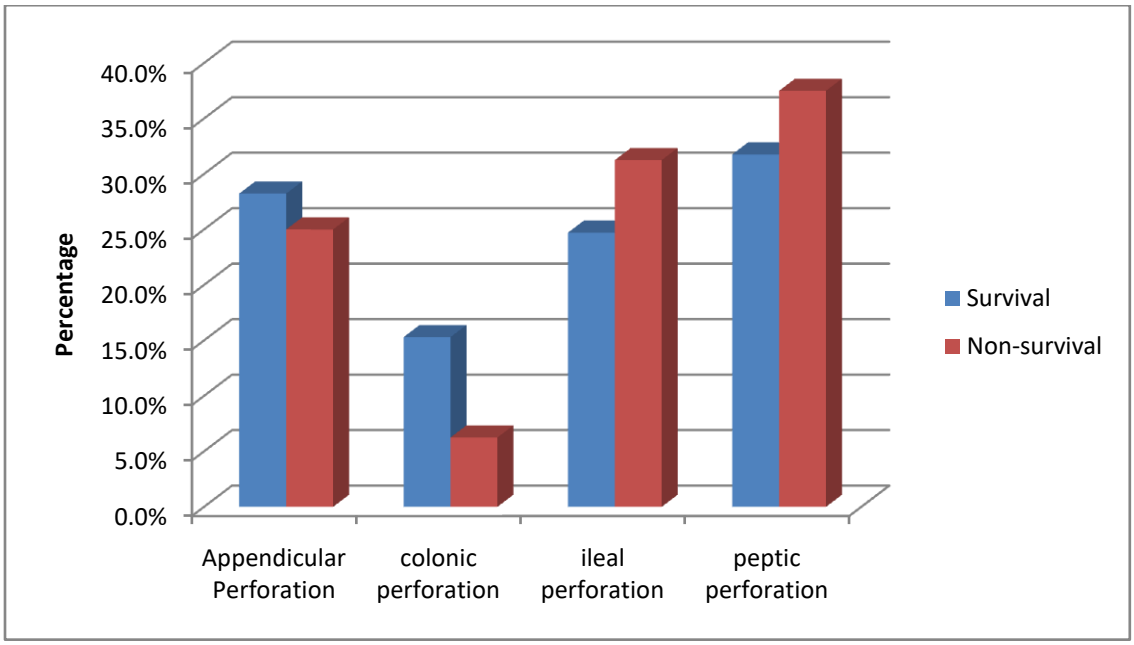

Figure 8: Shows the Mortality pattern based on site of perforation

9. Post operative Morbidity

Among 101 patients who presented with perforation peritonitis,14(13.9%) had wound infection,11 had intra-abdominal collection(10.9%),18 had respiratory complications(17.9%),6 had cardiac complications(5.9%),9 had acute renal failure(8.9%) and 3 had urinary tract infection(3%)

Table 9: Shows the distribution of patients according to post op morbidity

POST OP Morbidity	No. of cases	Percentage
1. Wound infection	14	13.9%
2. Intra abdominal collection	11	10.9%
3. Pneumonia or lung atelectasis	18	17.8%
4. Acute myocardial infarction or heart failure	6	5.9%
5. Acute renal failure	**9**	8.9%
6.Urinary tract infection	3	3.0%

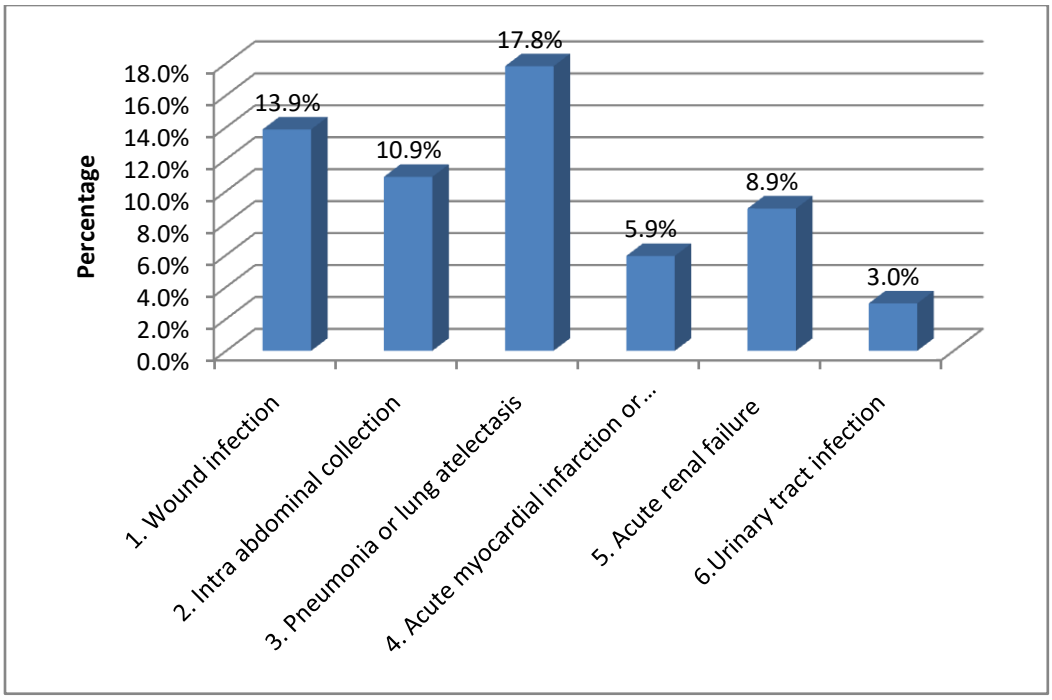

Figure 9: Shows the distribution of patients according to post op morbidity

10. Relation of post op morbidity with mortality

It has been observed in our study that 12(75%) patients out of 43 who had post operative morbidity were in the non survival group, The p value was<0.05 which was considered statistically significant.

Table 10: Relation of post morbidity with mortality

	MORTALITY	**Survival**		**Non-survival**		**Total**	**Chi-square value**	**p-value**
		Survival	**Survival**	**Non-survival**	**Non-survival**			
OVERALL MORBIDITY	NO	54	63.5%	4	25.0%	58	8.176	0.004
	YES	31	36.5%	12	75.0%	43		
Total		85	100.0%	16	100.0%	101		

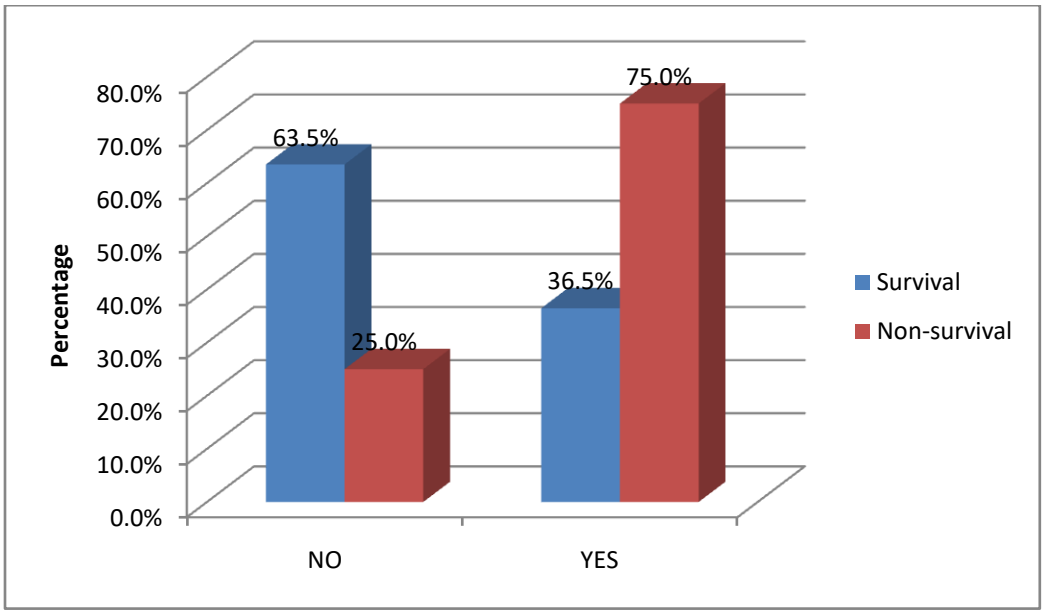

Figure 10: Relation of post op morbidity with mortality

11. Distribution of AEC between survival and non survival patients

Mean value of absolute eosinophil count at the the time of admission in the patients who survived was 18.03+/-65.32cells/$mm^3$and the mean value of absolute eosinophil count in the patients who didn't survive was 68.98+/-21.31cells/mm^3

Mean value of absolute eosinophil count after 24 hours of admission in patients who survived was 208.33+/-65.01cells/mm^3 and the mean value of patients who did not survive was 56 +/-17.97cells/mm^3

Mean value of absolute eosinophil count 72 hours after admission in patients who survived was 226.21+/-73.45cells/mm^3 and the mean value of patients who did not survive was 38.94 +/-12.57cells/mm^3

There was statistically significant difference in AEC between the two groups at the time of admission,24 hours after admission and 72 hours after admission. This shows that decrease in AEC, was associated with adverse outcome in perforation peritonitis patients with p value < 0.05 was statistically significant.

Table 11: Distribution of AEC between survival and non-survival patients

OVERALL MORTALITY	Survival		Non-survival		Z	p-value
	Mean	SD	Mean	SD		
At admission Absolute eosinophil COUNT	183.03	65.32	68.98	21.31	6.890	0.001
At 24 hours Absolute eosinophil count	208.33	65.01	56.00	17.97	9.271	0.001
At 72 hours Absolute eosinophil count	226.21	73.45	38.94	12.57	10.130	0.001
DIFF AT 24 HOURS	-25.30	27.93	12.98	11.80	-5.375	0.001
DIFF AT 72 HOURS	-43.18	59.09	30.04	14.91	-4.908	0.001

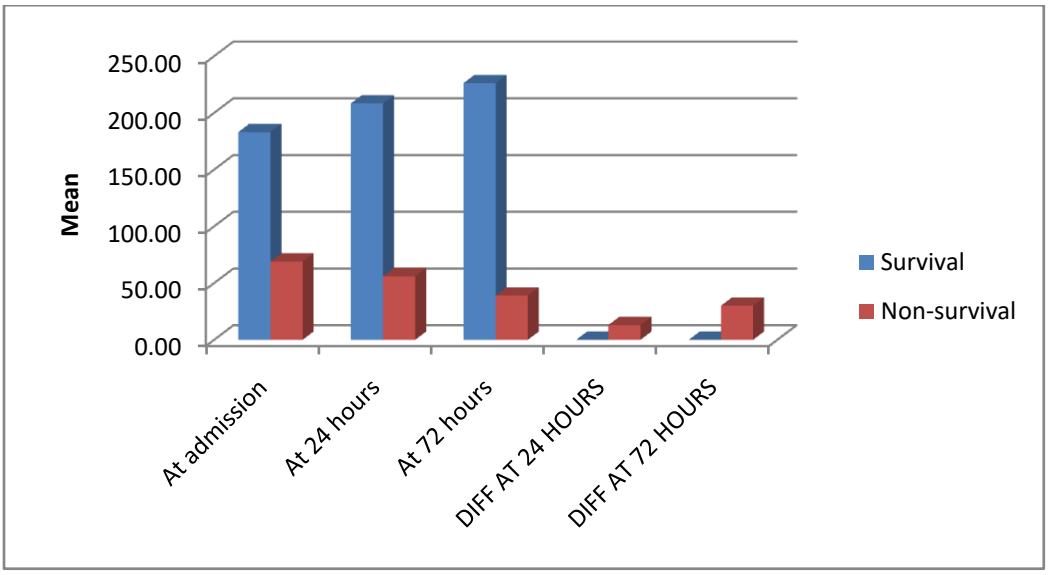

Figure 11: Distribution of AEC between survival and non-survival patients

12. Relation of AEC with survival and non survival group

In our study, mean value of absolute eosinophilic count in survival group was 205.84+/-63.11cells/mm^3 and in non-survival group was 54.69 +/16.25 cells/mm^3 with p value being less than 0.05 which is statistically significant.

Table 12: Relation of AEC with survival and non survival group

OVERALL MORTALITY	**Survival**		**Non-survival**		**Z**	**p-value**
	Mean	**SD**	**Mean**	**SD**		
ABSOLUTE EOSINOPHIL COUNT	205.84	63.11	54.69	16.25	9.485	0.001

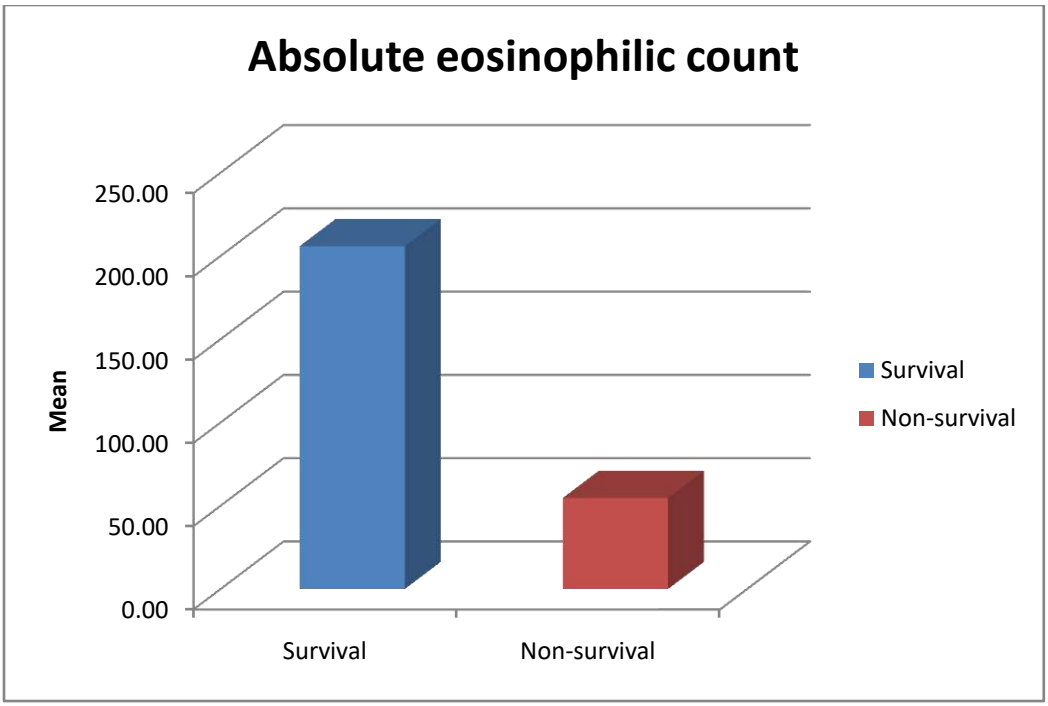

Figure 12: Relation of AEC with survival and non survival group

DISCUSSION

Perforation peritonitis is a very frequently encountered surgical emergency in tropical countries like India, most commonly affecting young men in their 4th or 5th decade of life. As far as management of the cases of perforation peritonitis is concerned , despite striking advances in various disciplines of medicine better understanding of etiopathology of disease and improved surgical technique, gastrointestinal perforation continues to remain of problem of high mortality and morbidity and complications.

In our study, the age group of the patients ranged from 18 years to 75 years with mean age being 49.53 with standard deviation of 18.54. In the study conducted by Swaminathan SP et al. [51] and Singh R et al [53]similar results were obtained with mean age being48.24+/-12.03and 40.04 ± 14.81 respectively. A similar finding was observed by Jain NK et al [4] in their study to assess the clinical profile and management of perforation peritionitis patients presenting to a tertiary care centre ,where majority of the patients were in 4th and 5th decade of life.

It was observed in our study that out of 101 patients,82(81.2%)were male and 19(18.8%) were females. The results were in accordance with the previous studies conducted by Swaminathan SP et al. [51](82.6% male and 17.3% females), Jagdeesh TS et al. [52](87.2% male and 12.7%female) and Prasad NBG and Reddy KVB [54](80% male and 20% female).This male preponderance can be explained by the fact that males are commonly involved in outdoor activities which make them more prone to gastrointestinal infections and trauma.

In our study, peptic perforation(32.7%) were the commonest, followed by appendicular (27.7%), Ileal perforation (25.7%) and colonic (13.9%) perfoation. In a study by Swaminathan SP et al. [51]it was found that the peptic (53%) perforations were commonest followed by ileal (39%), appendicular (5%) and colonic (3%) perforation. In a study done by

Jagdeesh TS et al. [52],similar results were obtained with peptic perforations being the commonest (58.5%) ,followed by ileal (36.1), colonic perforation(3.1%) and appendicular(2.1%) perforation. Various other studies done by Prasad NBG and Reddy KVB [54] and Singh R et al [53] similar results were seen. The perforations of proximal gastro–intestinal tract were more common in developing countries, which is in sharp contrast to developed countries where distal perforations were more common as shown in the study by Jain NK et al [4] to assess the clinical profile of patients with perforation peritonitis. This finding is consistent to the finding in our study which may be attributed to counter medication, smoking, and alcohol drinking being quite higher among males in this part of the country which in combination also increase the likelihood of perforation peritonitis.

In our study, out of 101 patients who presented with perforation peritonitis, 16(15.8%) patients were in mortality group and 85(84.2%) were in the survival group. Comparable to studies conducted by Swaminathan SP et al. [51]where out of 104 patients ,88(84.6%) patients were in the survival group and 16(18.8%) patients were in the mortality group, Jagdeesh TS et al. [52]where out of 94 patients,81(86.1%) patients were in the survival group and 13(13.8%) patients were in the mortality group and Singh R et al [53], where out of 84 patients 15 patients(17.86%) were in the non-survival group This is in contrast to the study conducted by Prasad NBG and Reddy KVB [54]where mortality rate was much higher i.e. 23.33%.

The age group of patients in our study ranged from 18 to 75 years, with a mean age of 49.53 and standard deviation of 18.54. Comparable results were found in studies conducted by Swaminathan SP et al. [51], Jagdeesh TS et al. [52], Prasad NBG and Reddy KVB [54], and Singh R et al [53]. Our study, as well as the previous studies, showed a higher proportion of males (81.2% in our study) in the patient population, which may be attributed to their increased involvement in outdoor activities and

subsequent susceptibility to gastrointestinal infections and trauma.

In our study, a total of 101 patients were analyzed, of which 22 had pre-existing co-morbidities. Among these co-morbidities, hypertension along with diabetes was the most common (27.2%), followed by hypertension (18.1%), diabetes mellitus (13.6%), coronary artery disease (9%), and asthma (4.5%) being the least common. Notably, 12 out of 16 non-survivor patients had pre-existing co-morbidities. A similar finding was reported in the study by Singh R et al.[53], where out of 15 patients who had mortality, 12 had pre-existing co-morbid conditions. These results suggest that pre-existing co-morbidities could contribute to increased mortality by adding stress.

In our study,16 patients who had a mortality,6(37.5%) were of peptic perforation, followed by ileal 5(31.3%), appendicular perforation 4(25%) and colonic perforation 1(6.3%).In the study by Singh et al out of 15 patients with mortality,9(60%) were of ileal in origin and 6(40%) were of duodenal in origin. In a study by Prasad NBG and Reddy KVB [54] 28 patients had mortality.11(39.2%) were of duodenal origin,3(10.7%) were of gastric origin,3(10.7%) were of small bowel in origin,3(10.7%) were of colonic and 1(3.5%) were of appendicular in origin.

Among the studied patients, respiratory-related complications (17.9%), wound infection (13.9%), and intra-abdominal collection (10.9%) were found to be the most common complications. Additionally, heart-related complications (5.9%), acute renal failure (8.9%), and urinary tract infection (3%) were also observed in some patients. These findings are consistent with those reported in previous studies by Prasad NBG and Reddy KVB [54] and Jhobhta RS et al[56]which identified respiratory-related complications as the most common complication, followed by wound infection, and abdominal collection.

The current study has demonstrated that among the 16 patients who had mortality, 12 patients had postoperative complications. This association between mortality and postoperative complications was found to be statistically significant, with a p-value of 0.001. These findings suggest that the presence of postoperative complications may increase the risk of mortality in patients. Therefore, it is crucial to identify and manage postoperative complications promptly to improve the outcomes of patients undergoing surgical interventions.

In a developing country like ours a majority of the cases present late in the hospital after taking conservative treatment from local practitioners and present with well-established generalized peritonitis with purulent / faecal contamination and varying degrees of septicemia. Assuming that these patients with perforation are septic upon admission, the determinants of mortality in sepsis should hold true for perforation peritonitis as well. Therefore , it is imperative to recognize patients at risk preoperatively and prepare for an intensive postoperative management strategy. This becomes more significant in our setup, where the intensive care facilities are limited and overwhelmed by the number of patients.

Many research and educational programs are being done at national and international level to improve the outcome of severe sepsis. On the other hand the developing countries are struggling in many ways to identify the patients as high risk and to treat them with intensive therapy since the resources are limited.

We observed in our study the association between the absolute eosinophil count (AEC) and clinical outcomes in patients with perforation peritonitis. The mean value of absolute eosinophil count at the time of admission was compared between patients who survived and those who did not. It was found that the mean absolute eosinophil count in patients who survived was 18.03+/-65.32, while the mean value for patients who did not survive was 68.98+/-21.31. After 24 hours of admission, the mean absolute eosinophil count in patients who survived was 208.33+/-65.01, whereas

the mean value for patients who did not survive was 56+/-17.97. Additionally, after 72 hours, the mean absolute eosinophil count in patients who survived was 226.21+/-73.45, while the mean value for patients who did not survive was 38.94+/-12.57.The mean value of absolute eosinophil count in patients in the survival group was 205.84+/-63.11 cells/mm^3 and the mean value in patients in the non survival group (mortality group) was 54.69 +/-16.25 cells/mm^3. In the study conducted by Swaminathan SP et al. [51] the mean value of AEC in patients in the survival group was 168.64+/-34.84 and in the non survival group was 33.13+/-7.50.In the study conducted by Jagdeesh TS et al. [52] the mean value of AEC in patients in the survival group was 73.4 +/-17.9 and in the non survival group was 35.5 +/-7.6. Our results were comparable to both the above mentioned studies.

Our study suggests that the decrease in AEC levels is an indication of poor prognosis as well as adverse outcomes in patients of perforation peritonitis. Therefore, AEC can be used as a reliable prognostic marker to assess the severity of the patient's condition in the hospital setting.

There are a very few studies available in the literature who have laid emphasis on the importance of AEC in prediciting morbidity and mortality of the patients with perforation peritonitis prospectively .Being a prospective study we could analyse the patients pre and post operative parameters in terms of patient variables which significantly contribute to the morbidity or mortality of the patient.

We are of the opinion that, AEC is a simple tool but promising tool in prognosticating patients with perforation peritonitis as it is part of the routine Complete blood count tests being routinely done for patients admitted in hospitals. It does not cause any extra effort or expenditure loss which is extremely relevant in a developing country like ours where majority of the population has financial constraints. Therefore, AEC can serve as a reliable prognostic marker to assess the severity of the patient's condition in a hospital setting.

SUMMARY AND CONCLUSION

The present study was conducted to study absolute eosinophilic count as a prognostic marker in cases of perforation peritonitis in 101 patients. After taking the consent, a detailed history and comprehensive general and local examination was carried out. The observations and results were tabulated and represented graphically. The summary of the observations is as follows.

- Majority of the patients were in the younger age group i.e. less than 30 years(23.8%),followed by patients with age between 51-60(22.8%).18 patients were in the age group >70 years(17.8%).14 patients were in the age group 61-70(13.9%)12 patients were in the age group 31-40(11.9%),And least were in the age group 41-50(9.9%).
- In our study there was male preponderance with number of males being 82(81.2%) and females being 19(18.8%).
- In our study, the most common site of perforation was peptic (n=33 ,32.7%) ,followed by appendicular (n=28,27.7%),followed by Ileal perforation (n=26,25.7%) and colonic (n=14,13.9%) was least among them.
- Out of 101 patients in our study who presented with perforation peritonitis mortality was seen in16(15.8%) patients .
- In our study, patients in the age group of more than 70 had the maximum number of mortalities i.e.5 out of 16 (31.3%).it was followed by 4 out of 16 (25%)patients in the age group of 31-40, 3 out of 16(18.8%) in the age group of 41-50,followed by 2 out of 16(12.5%) in the patients age less than 30 and 1 out 16 (6.3%) in the age group of 51-60 and 61-70 each.
- In our study, mortality was seen in 16 patients 14(87.5%) of which were males and 2(12.5%) were females
- In our study, out of 101 patients 22(21.78%) had pre existing co-

morbidities Asthma(1),Coronary artery disease(2),Diabetes mellitus(3),Typhoid(6),Hypertension(4) and Hypertension with Diabetes(6).Out of which 1(6.3%) with coronary artery disease had mortality,2(12.5%) with diabetic mellitus had mortality,2 (12.5%) with typhoid had mortality,1(6.3%) with hypertension had mortality and 3(18.8%) with both hypertension and diabetes had mortality.

- Out of 33 patients who presented with peptic perforation,6(37.5%) were in non survival group followed by ileal perforation of which 5(31.3%) out of 21 patients were in the non survival group, followed by appendicular perforation of which 4(25%) out of 28 patients were in the non survival group and 1(6.3%) out of 14 patients with colonic perforation who presented with perforation peritonitis were in the non-survival group.
- Among 101 patients who presented with perforation peritonitis,14(13.9%) had wound infection,11 had intra-abdominal collection(10.9%),18 had respiratory complications(17.9%),6 had heart related complications(5.9%),9 had acute renal failure(8.9%) and 3 had urinary tract infection(3%)
- It has been observed in our study that 12(75%) patients out of 43 who had post operative morbidity were in the non survival group, The p value was <0.05 which was considered statistically significant.
- Mean value of absolute eosinophil count at the the time of admission in the patients who survived was 18.03+/-65.32 cells/mm^3 and the mean value of absolute eosinophil count in the patients who didn't survive was 68.98+/-21.31cells/mm^3
- Mean value of absolute eosinophil count after 24 hours of admission in patients who survived was 208.33+/-65.01 cells/mm^3 and the mean value of patients who did not survive was 56 +/-17.97 cells/mm^3
- Mean value of absolute eosinophil count 72 hours after admission

in patients who survived was 226.21+/-73.45 cells/mm^3 and the mean value of patients who did not survive was 38.94 +/-12.57 cells/mm^3

- There was statistically significant difference in AEC between the two groups at the time of admission,24 hours after admission and 72 hours after admission. This shows that decrease in AEC, was associated with adverse outcome in perforation peritonitis patients. P value < 0.05 was statistically significant.
- In our study, mean value of absolute eosinophilic count in survival group was 205.84+/-63.11 cells/mm^3 and in non-survival group was 54.69 +/16.25 cells/mm^3 with p value being less than 0.05 which is statistically significant.

Conclusion

From this study, we conclude that AEC is a reliable marker of survival and it allows timely identification of high risk patients It can be used as a marker for risk stratification in perforation peritonitis patients. These findings may aid clinicians in the prompt identification and management of patients at risk for poor outcomes, thus improving the overall quality of care for patients with perforation peritonitis.

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PROFORMA

Sr. No:

NAME :	
AGE /SEX:	
ADDRESS WITH CONTACT NUMBER:	
MRD NO:	
DATE OF ADMISSION:	DATE OF SURGERY:

HISTORY

HISTORY OF PRESENTING ILLNESS

PAIN	**FEVER**
Origin:	Temperature:
Duration:	Duration :
Location:	Pattern:
Character:	

Signature of Candidate **Signature of Supervisor**

Any Other Relevant History	NAUSEA/ VOMITING Onset: Duration:

PAST HISTORY

Previous Surgery:

Previous Hospitalization:

Whether A Known Case Of DM/ TB/ Hypertension/
Asthma/ Epilepsy/ Cardiac Illness :

Signature of Candidate

Signature of Supervisor

CLINICAL EXAMINATION

GENERAL EXAMINATION	
APPEARANCE: VITALS: PR: BP: RR: Urine Output:	SENSORIUM:
SYSTEMIC EXAMINATION	
CVS:	RS:
PER ABDOMEN:	
Tenderness :	Guarding:
Rigidity:	Bowel sounds :

Signature of Candidate

Signature of Supervisor

INVESTIGATIONS:

HEMATOLOGICAL				
Date/ SNo.	TLC	DLC	AEC	PTI/INR

RFT ABG	LFT

Signature of Candidate **Signature of Supervisor**

RADIOLOGICAL INVESTIGATIONS
CXR: XRAY ABDOMEN: CECT ABDOMEN:

CLINICAL DIAGNOSIS:
SURGERY:

Signature of Candidate

Signature of Supervisor

POST OPERATIVE	
Wound infection Any 1 of the following: 1.Purulent wound discharge 2.Erythema >1cm from wound edges 3.Localised swelling pain or heat 4.Patient reporting increased tenderness Occurring within 30 days of Surgery	Intra- abdominal collection 1.Fever 2.TLC counts 3.Sugggestive CT findings
Pneumonia or lung atelectasis: 1.Xray changes 2..Respiratory rate 3.Blood urea nitrogen 4.CT changes	Acute myocardial infarction or heart failure: 1.ECG changes 2.Pain in chest 3.Troponon T levels.
Acute renal failure 1.Urine output 2.Creatinine 3.GFR	Urinary tract infection: 1.Fever (≥38°C) or chills. 2.New or increased burning pain on urination, frequency, or urgency. 3.New flank pain or suprapubic pain or tenderness
HOSPITAL STAY:	
AEC at the time of discharge:	
OVERALL MORBIDITY AND MORTALITY:	

Signature of Candidate **Signature of Supervisor**

KEY TO MASTER CHART

TLC	-	TOTAL LEUKOCYTE COUNT
N	-	NEUTROPHILS
L	-	LYMPHOCYTES
M	-	MONOCYTES
E	-	EOSINOPHILS
B	-	BASOPHILS
C SECTION	-	CAESAREAN SECTION
NSAIDS	-	NON STEROIDAL ANTI-INFLAMMATORY DRUGS

S.No	NAME	AGE	AGE GROUP	SEX	MRD NO	DATE OF ADMISSION	DATE OF SURGERY	HISTORY OF PRESENTING ILLNESS	Pain Orgin	Location	Nausea, Vomiting	Fever	Any other relevant history	MEDICAL	SURGICAL	DRUG	APPEARANCE	SENSORIUM	PR	SBP	DBP	RR
1	J	20	1	M	1239765	5-2-2021	5-2-2021	PAIN	Present	Epigastrium	Present	No	No	NIL	-	Yes,NSAIDS	fair	Alert	134	100	60	20
2	J	61	5	M	1264542	7-2-2021	7-2-2021	PAIN + NAUSEA	Present	RIF	Present	No	No	hypertensive	-	-	fair	Alert	102	100	60	18
3	KK	51	4	F	1155011	11-2-2021	11-2-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	Present	No	NIL	C SECTION	Yes,NSAIDS	sick	Alert	120	90	60	22
4	I	18	1	M	1152680	13-2-2021	13-2-2021	PAIN	Present	generalised	Present	No	No	NIL	-	-	fair	Alert	101	100	70	20
5	SS	71	6	M	1136354	16-2-2021	16-2-2021	PAIN + NAUSEA	Present	Epigastrium	Present	Present	No	NIL	-	Yes,NSAIDS	sick	Alert	128	80	56	24
6	RS	60	4	M	1078407	25-2-2021	25-2-2021	PAIN + NAUSEA	Present	Epigastrium	Present	No	No	NIL	-	Yes,NSAIDS	fair	Alert	102	90	60	20
7	R	42	3	M	1236713	2-3-2021	2-3-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	NIL	-	-	sick	Altered	134	76	52	24
8	LR	71	6	M	1185282	3-3-2021	3-3-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	Present	No	NIL	-	-	fair	Alert	112	100	56	22
9	Kk	40	2	F	1209475	5-3-2021	5-3-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	NIL	-	-	fair	Alert	102	96	60	18
10	SS	24	1	M	1086056	08/03/2021	3-8-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	No	No	NIL	-	-	fair	Alert	116	98	60	20
11	B	57	4	M	880977	11-3-2021	11-3-2021	PAIN	Present	generalised	Present	No	No	NIL	-	-	fair	Alert	108	100	64	18
12	MS	72	6	M	877375	15-3-2021	15-3-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	No	No	CAD	CHOLECYSTECTOMY	Yes,NSAIDS	sick	Alert	136	80	56	22
13	S	32	2	M	1174705	17-3-2021	17-3-2021	PAIN + NAUSEA	Present	Epigastrium	Present	Present	No	NIL	-	Yes,NSAIDS	sick	Alert	128	80	60	24
14	M	69	5	M	1243781	20-3-2021	21-3-2021	PAIN +NAUSEA + VOMITTING	Present	lower abdomen	Present	Present	No	NIL	-	-	sick	Alert	124	90	60	22
15	N	53	4	M	930473	21-3-2021	21-3-2021	PAIN +NAUSEA + VOMITTING	Present	lower abdomen	Present	No	No	NIL	-	-	fair	Alert	102	90	60	18
16	J	23	1	M	1161093	23-3-2021	23-3-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	Present	No	Hypertensive + DM	-	-	hippocratic facies	Altered	140	74	40	26
17	NS	70	5	M	1253300	28-3-2021	28-3-2021	PAIN	Present	Epigastrium	Present	Present	No	NIL	-	Yes,NSAIDS	fair	Alert	108	100	66	20
18	A	60	4	M	1227464	3-4-2021	3-4-2021	PAIN	Present	generalised	Present	No	No	NIL	-	-	fair	Alert	112	98	66	20
19	V	45	3	M	1208993	6-4-2021	6-4-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	DM	-	-	sick	Alert	132	86	56	24
20	A	23	1	M	1195762	8-4-2021	8-4-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	No	No	NIL	-	-	fair	Alert	118	100	60	18
21	A	36	2	M	1164575	12-4-2021	12-4-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	Present	No	NIL	-	-	sick	Alert	130	80	56	22
22	N	26	1	M	1100940	15-4-2021	15-4-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	Present	No	NIL	-	-	sick	Alert	126	90	60	20
23	AS	75	6	M	1881844	19-4-2021	19-4-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	No	No	NIL	-	-	fair	Alert	120	100	70	18
24	S	56	4	M	1015068	22-4-2021	22-4-2021	PAIN + NAUSEA	Present	lower abdomen	Present	Present	No	H/O TYPHOID	APPENDECTOMY	Yes,NSAIDS	sick	Alert	128	90	60	20
25	S	71	6	M	1136354	28-4-2021	28-4-2021	PAIN +NAUSEA + VOMITTING	Present	lower abdomen	Present	No	No	NIL	-	-	fair	Alert	114	104	74	18
26	D	35	2	M	1139954	3-5-2021	3-5-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	NIL	-	-	fair	Alert	122	96	66	20

S.No	Tenderness	Guarding	Rigidity	Bowel sounds	PER-RECTAL	TLC cells/mm3	N	L	M	E	B	At admission ABSOLUTE EOSINOPHILL COUNT	At 24 hours ABSOLUTE EOSINOPHILL COUNT	At 72 hours ABSOLUTE EOSINOPHILL COUNT	ABSOLUTE EOSINOPHILL COUNT	DIFF AT 24 HOURS	DIFF AT 72 HOURS	XRAY ABDOMEN	CECT ABDOMEN	CLINICAL DIAGNOSIS
1	present	present	present	sluggish	tenderness	18,000	80	15	4	1.2	0	216	234	202	217	-18	14	air under diaphragm	No	peptic perforation
2	present	present	present	sluggish	normal	16000	82	14	2.4	1	1	160	170	152	161	-10	8	air under diaphragm	No	appendicular perforation
3	present	present	present	nil	tenderness	21000	86	10	2	1	1	210	187	164	187	23	46	air under diaphragm	No	peptic perforation
4	present	present	present	sluggish	normal	15000	88	8	2	1	0	150	190	244	195	-40	-94	air under diaphragm	No	colonic perforation
5	present	present	present	nil	tenderness	4000	85	10	3	2	0	80	60	44	61	20	36	-	peptic perforation	peptic perforation
6	present	present	present	sluggish	normal	18000	82	13	2	2	1	360	400	423	394	-40	-63	air under diaphragm	No	peptic perforation
7	present	absent	present	nil	normal	22000	90	6	2.5	0.5	1	110	88	63	87	22	47	air under diaphragm	No	appendicular perforation
8	present	present	present	sluggish	normal	26000	88	7	3	1	1	260	330	392	327	-70	-132	air under diaphragm	No	ileal perforation
9	present	present	present	sluggish	normal	19000	80	16	3.3	1.4	0	266	216	176	219	50	90	air under diaphragm	No	appendicular perforation
10	present	present	present	sluggish	normal	17000	90	6	2	1	1	170	221	250	214	-51	-80	air under diaphragm	No	appendicular perforation
11	present	present	present	sluggish	normal	15000	88	7	3	2	0	300	324	388	337	-24	-88	air under diaphragm	No	ileal perforation
12	present	present	present	nil	tenderness	3600	80	15	2	2	1	72	45	24	47	27	48	air under diaphragm	No	peptic perforation
13	present	present	present	nil	tenderness	17000	89	9	1.4	0.6	0	102	154	198	151	-52	-96	air under diaphragm	No	peptic perforation
14	present	present	present	nil	tenderness	16000	84	12	3	1	0	160	202	223	195	-42	-63	air under diaphragm	No	colonic perforation
15	present	present	present	nil	normal	17000	90	6	2.2	0.8	1	136	174	202	171	-38	-66	air under diaphragm	No	appendicular perforation
16	present	absent	present	nil	tenderness	2800	90	6	2	2	0	56	36	25	39	20	31	air under diaphragm	No	peptic perforation
17	present	present	present	sluggish	normal	18000	86	10	3.1	1.1	0	198	220	192	203	-22	6	air under diaphragm	No	peptic perforation
18	present	present	present	sluggish	normal	15000	88	7	3	2	0	300	324	356	327	-24	-56	air under diaphragm	No	ileal perforation
19	present	present	present	nil	tenderness	20000	88	10	1.6	0.4	0	80	66	42	63	14	38	air under diaphragm	No	appendicular perforation
20	present	present	present	sluggish	normal	16000	82	14	2.4	1.6	1	256	264	238	253	-8	18	air under diaphragm	No	ileal perforation
21	present	present	present	nil	normal	19500	88	6	4.2	0.9	1	175	174	160	170	1	15	air under diaphragm	No	peptic perforation
22	present	present	present	nil	tenderness	17000	89	8	2	1	0	170	195	223	196	-25	-53	air under diaphragm	No	peptic perforation
23	present	present	present	sluggish	normal	18,000	80	15	4	1	0	180	202	234	205	-22	-54	air under diaphragm	No	peptic perforation
24	present	present	present	nil	tenderness	22000	90	6	2.5	0.9	1	198	212	196	202	-14	2	air under diaphragm	No	ileal perforation
25	present	present	present	sluggish	normal	16500	80	15	4	1	0	165	202	236	201	-37	-71	air under diaphragm	No	colonic perforation
26	present	present	present	sluggish	normal	17000	90	6	2	1	1	170	221	289	227	-51	-119	air under diaphragm	No	appendicular perforation

S.No	SURGERY	DURATION OF SURGERY (HRS)	POST OPMorbidity 1. Wound infection	2. Intra abdominal collection	3. Pneumonia or lung atelectasis	4. Acute myocardial infarction or heart failure	5. Acute renal failure	6. Urinary tract infection	HOSPITAL STAY (DAYS)	Absolute eosinophilic count at the time of discharge	OVERALL MORTALITY	OVERALL MORBIDITY
1	Modiied Graham's Patch repair with feeding jejunostomy	2	YES	NO	NO	NO	NO	NO	7	288	NO	YES
2	appendectomy	3	NO	YES	NO	NO	NO	NO	5	189	NO	YES
3	Modified Graham's Patch Repair	3	NO	NO	YES	NO	NO	NO	5	172	NO	YES
4	Resection anastomosis	2	NO	NO	NO	NO	NO	NO	4	298	NO	NO
5	Modiied Graham's Patch repair with feeding jejunostomy	4	NO	NO	YES	YES	NO	NO	8	-	YES	YES
6	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	NO	NO	NO	NO	NO	6	450	NO	NO
7	appendectomy	3	NO	NO	NO	YES	NO	NO	6	-	YES	YES
8	Resection anastomosis	4	NO	NO	NO	NO	NO	NO	5	402	NO	NO
9	appendectomy	4	NO	YES	NO	NO	NO	NO	6	184	NO	YES
10	appendectomy	2.5	NO	NO	NO	NO	NO	NO	6	289	NO	NO
11	resectiom with ileostomy	3	NO	NO	NO	NO	NO	NO	5	400	NO	NO
12	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	NO	YES	NO	YES	NO	7	-	YES	YES
13	Modified Graham's Patch Repair	2	NO	NO	NO	NO	NO	NO	8	202	NO	NO
14	perforation repair	2.5	NO	NO	NO	NO	NO	NO	7	242	NO	NO
15	appendectomy	3	NO	NO	NO	NO	NO	NO	5	276	NO	NO
16	Modified Graham's Patch Repair	5	NO	YES	NO	YES	NO	NO	7	-	YES	YES
17	Modiied Graham's Patch repair with feeding jejunostomy	4	YES	NO	NO	NO	NO	NO	9	210	NO	YES
18	Resection anastomosis	4	NO	NO	NO	NO	NO	NO	7	390	NO	NO
19	appendectomy	3	NO	NO	YES	NO	YES	NO	8	-	YES	YES
20	perforation repair and proximal illeostomy	3	NO	YES	NO	NO	NO	NO	8	250	NO	YES
21	Modiied Graham's Patch repair with feeding jejunostomy	4	NO	NO	YES	NO	NO	NO	9	200	NO	YES
22	Modiied Graham's Patch repair with feeding jejunostomy	3.5	NO	NO	NO	NO	NO	NO	7	250	NO	NO
23	Modified Graham's Patch Repair	2	NO	NO	NO	NO	NO	NO	5	290	NO	NO
24	Exploratory laparotomy with proximal loop ileostomy	3	NO	NO	YES	NO	NO	NO	13	210	NO	YES
25	repair and proximal colostomy	4	NO	NO	NO	NO	NO	NO	5	297	NO	NO
26	appendectomy	4	NO	NO	NO	NO	NO	NO	6	340	NO	NO

S.No	NAME	AGE	AGE GROUP	SEX	MRD NO	DATE OF ADMISSION	DATE OF SURGERY	HISTORY OF PRESENTING ILLNESS	Pain Orgin	Location	Nausea, Vomiting	Fever	Any other relevant history	MEDICAL	SURGICAL	DRUG	APPEARANCE	SENSORIUM	PR	SBP	DBP	RR
27	G	18	1	M	1234300	5-5-2021	5-5-2021	PAIN	Present	generalised	Present	Present	No	NIL	-	-	fair	Alert	118	100	60	18
28	A	18	1	M	1229447	10-5-2021	10-5-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	NIL	-	-	fair	Alert	120	102	70	18
29	R	28	1	M	1176067	13-5-2021	13-5-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	Present	No	NIL	-	Yes,NSAIDS	sick	Alert	134	80	60	20
30	R	49	3	M	1163567	16-5-2021	16-5-2021	PAIN + NAUSEA	Present	Epigastrium	Present	No	No	NIL	-	-	fair	Alert	124	96	60	20
31	TK	60	4	F	1201043	20-5-2021	20-5-2021	PAIN +NAUSEA + VOMITTING	Present	lower abdomen	Present	Present	No	NIL	-	-	sick	Alert	126	80	56	22
32	H	25	1	M	1203338	22-5-2021	22-5-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	Hypertensive + DM	-	-	sick	Altered	136	74	40	24
33	B	75	6	M	1141875	25-5-2021	25-5-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	asthma	-	-	sick	Alert	130	90	60	20
34	S	48	3	M	1202459	1-6-2021	1-6-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	NIL	-	-	fair	Alert	120	102	70	16
35	CK	60	4	F	1253181	3-6-2021	3-6-2021	PAIN + NAUSEA	Present	Epigastrium	Present	Present	No	NIL	CHOLECYSTECTOMY	Yes,NSAIDS	sick	Alert	132	80	60	22
36	SK	35	2	M	1266788	6-6-2021	6-6-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	No	No	NIL	-	Yes,NSAIDS	fair	Alert	112	104	70	18
37	AS	37	2	M	1280904	9-6-2021	9-6-2021	PAIN +NAUSEA + VOMITTING	Present	lower abdomen	Present	No	No	H/O TYPHOID	-	-	fair	Alert	122	90	66	20
38	AD	75	6	F	1284907	11-6-2021	11-6-2021	PAIN	Present	generalised	Present	Present	No	Hypertensive + DM	-	-	fair	Alert	126	92	60	20
39	CS	36	2	M	1285451	13-6-2021	13-6-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	Present	No	NIL	-	Yes,NSAIDS	sick	Alert	138	74	40	22
40	UR	74	6	F	1286236	15-6-2021	15-6-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	No	No	NIL	-	-	fair	Alert	124	100	60	20
41	P	55	4	M	978972	17-6-2021	18-6-2021	PAIN	Present	lower abdomen	Present	Present	No	H/O TYPHOID	-	-	sick	Alert	132	80	56	20
42	JS	52	4	F	1288286	22-6-2021	22-6-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	Present	No	hypertensive	C SECTION	-	sick	Altered	140	74	40	26
43	AS	42	3	M	1290957	25-6-2021	25-6-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	No	No	NIL	-	-	fair	Alert	116	94	66	20
44	NS	28	1	M	1299609	2-7-2021	2-7-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	NIL	-	-	sick	Alert	122	90	60	22
45	GK	75	6	F	1300302	8-7-2021	8-7-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	Present	No	NIL	-	-	fair	Alert	112	100	60	20
46	VB	50	3	M	1300241	10-7-2021	10-7-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	Present	No	NIL	INGUINAL HERNIA	Yes,NSAIDS	fair	Alert	114	98	56	20
47	S	22	1	M	898423	14-7-2021	14-7-2021	PAIN + NAUSEA	Present	Epigastrium	Present	No	No	NIL	-	-	fair	Alert	104	102	60	18
48	HK	23	1	F	1309725	18-7-2021	18-7-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	NIL	-	-	sick	Alert	134	80	56	22
49	DS	59	4	M	462811	22-7-2021	22-7-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	Present	No	NIL	-	-	fair	Alert	110	112	68	20
50	KK	71	6	M	1330159	28-7-2021	28-7-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	No	No	NIL	-	Yes,NSAIDS	fair	Alert	118	90	60	20

S.No	Tenderness	Guarding	Rigidity	Bowel sounds	PER-RECTAL	TLC cells/mm3	N	L	M	E	B	At admission ABSOLUTE EOSINOPHILL COUNT	At 24 hours ABSOLUTE EOSINOPHILL COUNT	At 72 hours ABSOLUTE EOSINOPHILL COUNT	ABSOLUTE EOSINOPHILL COUNT	DIFF AT 24 HOURS	DIFF AT 72 HOURS	XRAY ABDOMEN	CECT ABDOMEN	CLINICAL DIAGNOSIS
27	present	present	present	sluggish	normal	15000	88	7	3	2	0	300	324	388	337	-24	-88	air under diaphragm	No	ileal perforation
28	present	present	present	sluggish	normal	16000	84	12	3	1	0	160	202	256	206	-42	-96	air under diaphragm	No	appendicular perforation
29	present	present	present	nil	tenderness	19500	88	6	4.2	0.8	1	156	102	88	115	54	68	air under diaphragm	No	peptic perforation
30	present	present	present	sluggish	normal	14000	82	14	3	1	0	140	179	224	181	-39	-84	air under diaphragm	No	peptic perforation
31	present	present	present	nil	normal	15000	83	13	3	1	0	150	194	172	172	-44	-22	air under diaphragm	No	ileal perforation
32	present	absent	present	nil	tenderness	4600	84	10	4	1	1	46	38	36	40	8	10	air under diaphragm	No	appendicular perforation
33	present	present	present	nil	tenderness	18,000	80	15	4	1	0	180	202	234	205	-22	-54	-	appendicular perforation	appendicular perforation
34	present	present	present	sluggish	normal	17000	89	8	2	1.2	0	204	223	195	207	-19	9	air under diaphragm	No	appendicular perforation
35	present	present	present	nil	tenderness	16000	82	14	2.4	1.8	1	288	280	246	271	8	42	air under diaphragm	No	peptic perforation
36	present	present	present	sluggish	normal	17000	90	6	2.2	0.8	1	136	174	243	184	-38	-107	air under diaphragm	No	peptic perforation
37	present	present	present	sluggish	normal	6000	87	10	2.1	0.9	0	54	66	41	54	-12	13	air under diaphragm	No	ileal perforation
38	present	present	present	sluggish	normal	16000	82	14	2.4	1.4	1	224	234	228	229	-10	-4	air under diaphragm	No	ileal perforation
39	present	present	present	nil	tenderness	22000	87	10	2.6	0.4	0	88	72	44	68	16	44	air under diaphragm	No	peptic perforation
40	present	present	present	sluggish	normal	14500	83	12	4	1.6	0	232	240	190	221	-8	42	air under diaphragm	No	appendicular perforation
41	present	present	present	nil	tenderness	5000	88	8	2	2	0	100	157	192	150	-57	-92	air under diaphragm	No	ileal perforation
42	present	present	present	nil	normal	19500	88	6	4.2	0.8	1	156	112	86	118	44	70	air under diaphragm	No	ileal perforation
43	present	present	present	sluggish	normal	14000	82	14	3	1	0	140	179	224	181	-39	-84	air under diaphragm	No	ileal perforation
44	present	present	present	sluggish	normal	18,000	80	15	4	1	0	180	202	234	205	-22	-54	air under diaphragm	No	appendicular perforation
45	present	present	present	sluggish	normal	15000	88	7	3	2	0	300	324	336	320	-24	-36	air under diaphragm	No	peptic perforation
46	present	present	present	sluggish	normal	17200	88	6	4.2	0.8	1	137.6	186	218	181	-48.4	-80.4	air under diaphragm	No	peptic perforation
47	present	present	present	sluggish	normal	18,000	80	15	4	1	0	180	202	248	210	-22	-68	air under diaphragm	No	peptic perforation
48	present	present	present	nil	tenderness	16000	82	14	2.4	0.6	1	96	156	189	147	-60	-93	air under diaphragm	No	appendicular perforation
49	present	present	present	sluggish	normal	15000	88	7	3	2	0	300	324	340	321	-24	-40	air under diaphragm	No	peptic perforation
50	present	present	present	sluggish	normal	18,000	80	15	4	1	0	180	202	234	205	-22	-54	air under diaphragm	No	peptic perforation

S.No	SURGERY	DURATION OF SURGERY (HRS)	POST OPMorbidity 1. Wound infection	2. Intra abdominal collection	3. Pneumonia or lung atelectasis	4. Acute myocardial infarction or heart failure	5. Acute renal failure	6. Urinary tract infection	HOSPITAL STAY (DAYS)	Absolute eosinophilic count at the time of discharge	OVERALL MORTALITY	OVERALL MORBIDITY
27	Resection anastomosis	3.5	NO	NO	NO	NO	NO	NO	6	402	NO	NO
28	appendectomy	3	NO	NO	NO	NO	NO	NO	6	279	NO	NO
29	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	YES	NO	NO	YES	NO	9	102	NO	YES
30	Modified Graham's Patch Repair	4	YES	NO	NO	NO	NO	NO	8	260	NO	YES
31	Resection anastomosis	3	NO	YES	NO	NO	NO	NO	10	158	NO	YES
32	appendectomy	4	NO	NO	YES	NO	NO	NO	5	-	YES	YES
33	appendectomy	3.5	NO	NO	NO	NO	NO	NO	6	276	NO	NO
34	appendectomy	3	YES	NO	NO	NO	NO	NO	9	220	NO	YES
35	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	YES	NO	NO	NO	NO	11	220	NO	YES
36	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	NO	NO	NO	NO	NO	9	297	NO	NO
37	repair and proximal colostomy	4	NO	NO	YES	NO	NO	NO	8	-	YES	YES
38	Resection anastomosis	4	NO	YES	NO	NO	NO	NO	12	240	NO	YES
39	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	NO	YES	NO	NO	NO	7	-	YES	YES
40	appendectomy	5	YES	NO	NO	NO	NO	YES	11	160	NO	YES
41	Resection anastomosis	3	NO	NO	NO	NO	NO	NO	6	248	NO	NO
42	Resection anastomosis	3	NO	NO	YES	NO	NO	NO	12	92	NO	YES
43	repair and proximal colostomy	2.5	NO	NO	NO	NO	NO	NO	6	273	NO	NO
44	appendectomy	4.5	NO	NO	NO	NO	NO	NO	7	290	NO	NO
45	Modiied Graham's Patch repair with feeding jejunostomy	4	NO	NO	NO	NO	NO	NO	7	350	NO	NO
46	Modiied Graham's Patch repair with feeding jejunostomy	3	YES	NO	NO	NO	NO	NO	11	210	NO	YES
47	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	NO	NO	NO	NO	NO	8	296	NO	NO
48	appendectomy	4	NO	NO	NO	NO	NO	YES	10	178	NO	YES
49	Modiied Graham's Patch repair with feeding jejunostomy	2.5	NO	NO	NO	NO	NO	NO	9	370	NO	NO
50	Modiied Graham's Patch repair with feeding jejunostomy	3.5	NO	NO	NO	NO	NO	NO	8	298	NO	NO

S.No	NAME	AGE	AGE GROUP	SEX	MRD NO	DATE OF ADMISSION	DATE OF SURGERY	HISTORY OF PRESENTING ILLNESS	Pain Orgin	Location	Nausea, Vomiting	Fever	Any other relevant history	MEDICAL	SURGICAL	DRUG	APPEARANCE	SENSORIUM	PR	SBP	DBP	RR
51	TS	30	1	M	986526	3-8-2021	3-8-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	No	No	NIL	-	Yes,NSAIDS	fair	Alert	120	92	60	20
52	H	26	1	M	1332490	7-8-2021	7-8-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	No	No	NIL	-	-	sick	Alert	120	80	60	22
53	SS	34	2	M	1333425	14-8-2021	15-8-2021	PAIN + NAUSEA	Present	generalised	Present	Present	No	DM	-	-	sick	Alert	128	80	56	24
54	KS	18	1	M	945991	19-8-2021	19-8-2021	PAIN + NAUSEA	Present	lower abdomen	Present	Present	No	H/O TYPHOID	-	-	fair	Alert	118	88	54	20
55	KS	70	5	M	1322647	27-8-2021	27-8-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	No	No	NIL	-	Yes,NSAIDS	fair	Alert	130	80	54	22
56	CK	74	6	F	1389139	3-9-2021	3-9-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	No	No	NIL	HYSTERECTOMY	Yes,NSAIDS	sick	Alert	130	72	40	24
57	AM	35	2	M	1127632	7-9-2021	7-9-2021	PAIN	Present	generalised	Present	Present	No	NIL	-	Yes,NSAIDS	sick	Alert	132	80	56	22
58	R	30	1	M	1282292	11-9-2021	11-9-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	No	No	NIL	-	-	fair	Alert	116	100	60	18
59	JS	40	2	M	1392931	14-9-2021	14-9-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	Present	No	hypertensive	-	-	sick	Alert	138	74	40	24
60	GS	53	4	M	894094	19-9-2021	19-9-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	No	No	NIL	-	-	fair	Alert	124	92	60	20
61	SD	72	6	F	896329	24-9-2021	24-9-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	No	No	NIL	-	-	fair	Alert	114	96	60	18
62	MS	65	5	M	1396408	28-9-2021	28-9-2021	PAIN	Present	Epigastrium	Present	No	No	hypertensive	-	Yes,NSAIDS	fair	Alert	102	100	60	20
63	DS	63	5	M	1398117	3-10-2021	3-10-2021	PAIN	Present	generalised	Present	Present	No	NIL	-	-	fair	Alert	130	100	60	18
64	M K	72	6	F	939636	7-10-2021	7-10-2021	PAIN	Present	Epigastrium	Present	No	No	NIL	C SECTION	-	sick	Alert	112	90	60	22
65	P	60	4	M	1332922	13-10-2021	13-10-2021	PAIN + NAUSEA	Present	generalised	Present	Present	No	NIL	-	Yes,NSAIDS	fair	Alert	110	100	70	20
66	RS	56	4	M	1332607	18-10-2021	18-10-2021	PAIN	Present	generalised	Present	No	No	NIL	-	-	fair	Alert	102	80	56	20
67	J	72	6	M	231183	22-10-2021	22-10-2021	PAIN + NAUSEA	Present	generalised	Present	Present	No	Hypertensive + DM	-	-	sick	Altered	146	90	60	24
68	BK	56	4	F	1370195	30-10-2021	30-10-2021	PAIN	Present	lower abdomen	Present	Present	No	NIL	-	-	sick	Alert	138	76	52	24
69	B	50	3	M	1386419	5-11-2021	5-11-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	NIL	-	-	fair	Alert	106	100	56	22
70	BS	69	5	M	1330146	11-11-2021	11-11-2021	PAIN +NAUSEA + VOMITTING	Present	lower abdomen	Present	No	No	NIL	-	-	sick	Alert	110	96	60	18
71	RG	47	3	M	818886	15-11-2021	15-11-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	No	No	NIL	-	-	sick	Alert	100	98	60	20
72	JS	30	1	M	876658	24-11-2021	24-11-2021	PAIN +NAUSEA + VOMITTING	Present	lower abdomen	Present	No	No	CAD	-	-	fair	Alert	108	100	64	18
73	BR	62	5	M	1413242	30-11-2021	30-11-2021	PAIN + NAUSEA	Present	generalised	Present	Present	No	NIL	-	-	sick	Alert	118	80	56	22
74	R	57	4	F	9209210	4-12-2021	4-12-2021	PAIN	Present	generalised	Present	Present	No	NIL	-	-	sick	Alert	112	80	60	24
75	BS	26	1	M	1440276	10-12-2021	10-12-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	No	No	NIL	-	Yes,NSAIDS	sick	Alert	120	90	60	22
76	SG	57	4	F	1348447	16-12-2021	16-12-2021	PAIN + NAUSEA	Present	lower abdomen	Present	Present	No	NIL	CHOLECYSTECTOMY	-	fair	Alert	114	90	60	18

S.No	Tenderness	Guarding	Rigidity	Bowel sounds	PER-RECTAL	TLC cells/mm3	N	L	M	E	B	At admission ABSOLUTE EOSINOPHILL COUNT	At 24 hours ABSOLUTE EOSINOPHILL COUNT	At 72 hours ABSOLUTE EOSINOPHILL COUNT	ABSOLUTE EOSINOPHILL COUNT	DIFF AT 24 HOURS	DIFF AT 72 HOURS	XRAY ABDOMEN	CECT ABDOMEN	CLINICAL DIAGNOSIS
51	present	present	present	sluggish	normal	17000	89	8	2	1	0	170	195	223	196	-25	-53	air under diaphragm	No	appendicular perforation
52	present	present	present	sluggish	normal	19500	88	6	4.2	0.8	1	156	102	92	117	54	64	air under diaphragm	No	appendicular perforation
53	present	present	present	nil	tenderness	20000	88	10	1.6	0.4	0	80	66	42	63	14	38	air under diaphragm	No	ileal perforation
54	present	present	present	sluggish	normal	5500	88	8	3.3	0.7	0	38.5	44	88	57	-5.5	-49.5	air under diaphragm	No	ileal perforation
55	present	present	present	sluggish	normal	18,000	80	15	4	1	0	180	224	278	227	-44	-98	air under diaphragm	No	peptic perforation
56	present	present	present	sluggish	normal	24000	82	14	3	0.7	0.3	168	172	102	147	-4	66	air under diaphragm	No	peptic perforation
57	present	present	present	sluggish	normal	16000	82	14	2.4	0.6	1	96	134	156	129	-38	-60	air under diaphragm	No	ileal perforation
58	present	present	present	sluggish	normal	15000	88	7	3	2	0	300	316	302	306	-16	-2	air under diaphragm	No	appendicular perforation
59	present	present	present	nil	tenderness	2800	90	6	2	2	0	56	36	25	39	20	31	air under diaphragm	No	ileal perforation
60	present	present	present	sluggish	normal	16500	80	15	4	1	0	165	202	236	201	-37	-71	air under diaphragm	No	ileal perforation
61	present	present	present	sluggish	normal	17000	90	6	2	1	1	170	221	312	234	-51	-142	air under diaphragm	No	appendicular perforation
62	present	present	present	sluggish	normal	15000	88	8	2	1	0	150	190	244	195	-40	-94	air under diaphragm	No	peptic perforation
63	present	present	present	sluggish	normal	16000	82	14	2.4	0.6	1	96	134	178	136	-38	-82	air under diaphragm	No	ileal perforation
64	present	present	present	nil	tenderness	4800	86	10	3.8	1.2	1	57.6	50	42	50	7.6	15.6	air under diaphragm	No	peptic perforation
65	present	present	present	sluggish	normal	18000	88	8	2	1	0	150	190	244	195	-40	-94	air under diaphragm	No	colonic perforation
66	present	present	present	nil	normal	21000	86	10	2	1	1	210	182	162	185	28	48	air under diaphragm	No	colonic perforation
67	present	absent	present	sluggish	normal	22000	90	6	2.5	0.5	1	110	88	66	88	22	44	air under diaphragm	No	appendicular perforation
68	present	present	present	nil	tenderness	19000	88	8	2.5	0.5	1	95	118	156	123	-23	-61	air under diaphragm	No	ileal perforation
69	present	present	present	sluggish	normal	26000	88	7	3	1	1	260	330	392	327	-70	-132	air under diaphragm	No	appendicular perforation
70	present	present	present	sluggish	tenderness	19000	80	16	3.3	1.1	0	209	226	163	199	-17	46	air under diaphragm	No	ileal perforation
71	present	present	present	sluggish	normal	17000	90	6	2	1	1	170	210	239	206	-40	-69	air under diaphragm	No	appendicular perforation
72	present	present	present	sluggish	normal	15000	88	7	3.6	1.4	0	210	274	324	269	-64	-114	-	Appendiceal Perforation	Appendiceal Perforation
73	present	present	present	nil	normal	3600	80	15	2	2	1	72	45	24	47	27	48	air under diaphragm	No	ileal perforation
74	present	present	present	nil	tenderness	17000	89	9	1.4	1	0	170	188	130	163	-18	40	air under diaphragm	No	peptic perforation
75	present	present	present	nil	normal	17000	90	6	2.2	0.8	1	136	174	202	171	-38	-66	air under diaphragm	No	peptic perforation
76	present	present	present	nil	normal	16000	84	12	3	1	0	160	202	223	195	-42	-63	air under diaphragm	No	colonic perforation

S.No	SURGERY	DURATION OF SURGERY (HRS)	POST OPMorbidity 1. Wound infection	2. Intra abdominal collection	3. Pneumonia or lung atelectasis	4. Acute myocardial infarction or heart failure	5. Acute renal failure	6. Urinary tract infection	HOSPITAL STAY (DAYS)	Absolute eosinophilic count at the time of discharge	OVERALL MORTALITY	OVERALL MORBIDITY
51	appendectomy	4	NO	NO	NO	NO	NO	NO	8	273	NO	NO
52	appendectomy	3	NO	YES	NO	NO	YES	NO	15	76	NO	YES
53	perforation repair and proximal illeostomy	4	NO	YES	YES	NO	NO	NO	13	-	YES	YES
54	perforation repair and proximal illeostomy	3	NO	YES	NO	NO	NO	NO	9	92	NO	YES
55	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	NO	NO	NO	NO	NO	7	307	NO	NO
56	Modiied Graham's Patch repair with feeding jejunostomy	3	YES	NO	NO	NO	NO	NO	7	104	NO	YES
57	perforation repair and proximal illeostomy	5	NO	NO	NO	NO	NO	NO	7	197	NO	NO
58	appendectomy	2	NO	NO	NO	NO	NO	NO	9	359	NO	NO
59	Resection anastomosis	3	NO	NO	YES	NO	NO	NO	12	-	YES	YES
60	Resection anastomosis	3.5	NO	NO	NO	NO	NO	NO	8	256	NO	NO
61	appendectomy	4	NO	NO	NO	NO	NO	NO	8	330	NO	NO
62	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	NO	NO	NO	NO	NO	5	289	NO	NO
63	Exploratory laparotomy with proximal loop ileostomy	5	NO	NO	NO	NO	NO	NO	6	245	NO	NO
64	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	NO	NO	NO	NO	NO	6	-	YES	NO
65	Hartmann's Procedure	3	NO	NO	NO	NO	NO	NO	7	312	NO	NO
66	repair and proximal colostomy	2.5	NO	NO	NO	NO	NO	NO	7	175	NO	YES
67	appendectomy	4.5	NO	NO	YES	YES	YES	NO	4	-	YES	YES
68	Exploratory laparotomy with proximal loop ileostomy	4	NO	NO	NO	NO	NO	NO	7	198	NO	NO
69	appendectomy	3	NO	NO	NO	NO	NO	NO	6	412	NO	NO
70	perforation repair and proximal illeostomy	3	NO	NO	YES	NO	YES	NO	14	189	NO	YES
71	appendectomy	4	NO	NO	NO	NO	NO	NO	8	289	NO	NO
72	appendectomy	2.5	NO	NO	NO	NO	NO	NO	6	356	NO	NO
73	perforation repair and proximal illeostomy	3.5	NO	NO	NO	NO	NO	NO	8	-	YES	NO
74	Modiied Graham's Patch repair with feeding jejunostomy	4	YES	NO	NO	NO	NO	NO	7	189	NO	YES
75	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	NO	NO	NO	NO	NO	6	234	NO	NO
76	Exploratory laparotomy with proximal loop ileostomy	4	NO	NO	NO	NO	NO	NO	5	278	NO	NO

S.No	NAME	AGE	AGE GROUP	SEX	MRD NO	DATE OF ADMISSION	DATE OF SURGERY	HISTORY OF PRESENTING ILLNESS	Pain Orgin	Location	Nausea, Vomiting	Fever	Any other relevant history	MEDICAL	SURGICAL	DRUG	APPEARANCE	SENSORIUM	PR	SBP	DBP	RR
77	SP	55	4	M	1166496	24-12-2021	24-12-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	Present	No	NIL	-	-	hippocratic facies	Altered	142	72	40	26
78	R	60	4	M	1151879	31-12-2021	31-12-2021	PAIN	Present	generalised	Present	No	No	NIL	-	-	fair	Alert	108	100	66	20
79	J	20	1	M	1158992	5-1-2022	5-1-2022	PAIN	Present	RIF	Present	Present	No	DM	-	-	fair	Alert	110	98	66	20
80	GS	70	5	M	1158992	17-1-2022	17-1-2022	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	No	No	NIL	-	-	sick	Alert	130	86	56	24
81	H	25	1	M	1202169	25-1-2022	25-1-2022	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	Present	No	NIL	-	-	fair	Alert	106	100	60	18
82	M	61	5	M	1130063	8-2-2022	8-2-2022	PAIN + NAUSEA	Present	Epigastrium	Present	Present	No	NIL	-	Yes,NSAIDS	sick	Alert	124	80	56	22
83	GK	52	4	F	904036	14-2-2022	14-2-2022	PAIN	Present	generalised	Present	No	No	NIL	-	-	sick	Alert	112	90	60	20
84	GS	40	2	M	1392929	21-2-2021	21-2-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	Present	No	H/O TYPHOID	-	-	fair	Alert	110	100	70	18
85	RM	19	1	M	1323343	28-2-2021	28-2-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	No	No	NIL	-	Yes,NSAIDS	sick	Alert	128	90	60	20
86	JS	72	6	M	1394776	5-3-2021	5-3-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	Present	No	NIL	-	-	fair	Alert	111	104	74	18
87	NS	62	5	M	1333749	11-3-2021	11-3-2021	PAIN +NAUSEA + VOMITTING	Present	Umbilical	Present	Present	No	NIL	-	-	fair	Alert	110	96	66	20
88	KS	75	6	M	1308709	18-3-2021	18-3-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	Present	No	NIL	-	-	fair	Alert	128	100	60	18
89	HS	70	5	M	918109	27-3-2021	27-3-2021	PAIN	Present	generalised	Present	Present	No	NIL	-	-	sick	Alert	110	102	70	18
90	RA	75	6	M	1391971	4-4-2021	4-4-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	No	No	NIL	APPENDECTOMY	Yes,NSAIDS	sick	Alert	116	80	60	20
91	S	48	3	M	919278	12-4-2021	12-4-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	Present	No	NIL	-	-	fair	Alert	108	96	60	20
92	RK	52	4	M	1151768	23-4-2021	23-4-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	Present	No	Hypertensive + DM	-	-	sick	Alert	122	80	56	22
93	B	72	6	F	875531	10-5-2021	10-5-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	Present	No	NIL	INCISIONAL HERNIA OPEN REPAIR	Yes,NSAIDS	sick	Altered	138	74	40	24
94	NK	60	4	M	1172291	19-5-2021	19-5-2021	PAIN +NAUSEA + VOMITTING	Present	generalised	Present	Present	No	NIL	-	-	sick	Alert	114	90	60	20
95	P	68	5	M	1413532	1-6-2021	1-6-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	Present	No	NIL	-	Yes,NSAIDS	fair	Alert	106	102	70	16
96	AK	55	4	F	1406515	12-6-2021	12-6-2021	PAIN +NAUSEA + VOMITTING	Present	Epigastrium	Present	No	No	NIL	-	Yes,NSAIDS	sick	Alert	112	80	60	22
97	SS	49	3	M	1256840	22-6-2021	22-6-2021	PAIN + NAUSEA	Present	lower abdomen	Present	No	No	H/O TYPHOID	CHOLECYSTECTOMY	-	sick	Alert	120	90	50	24
98	SS	65	5	M	1332504	7-7-2021	7-7-2021	PAIN	Present	generalised	Present	Present	No	Hypertensive + DM	-	-	fair	Alert	112	90	66	20
99	JS	38	2	M	1287371	12-7-2021	12-7-2021	PAIN +NAUSEA + VOMITTING	Present	lower abdomen	Present	Present	No	NIL	-	-	fair	Alert	126	92	60	18
100	S	22	1	F	1020113	18-7-2021	18-7-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	No	No	NIL	-	-	sick	Alert	144	74	40	20
101	BS	18	1	M	1111466	28-7-2021	28-7-2021	PAIN +NAUSEA + VOMITTING	Present	RIF	Present	No	No	NIL	-	-	fair	Alert	104	100	60	16

S.No	Tenderness	Guarding	Rigidity	Bowel sounds	PER-RECTAL	TLC cells/mm3	N	L	M	E	B	At admission ABSOLUTE EOSINOPHILL COUNT	At 24 hours ABSOLUTE EOSINOPHILL COUNT	At 72 hours ABSOLUTE EOSINOPHILL COUNT	ABSOLUTE EOSINOPHILL COUNT	DIFF AT 24 HOURS	DIFF AT 72 HOURS	XRAY ABDOMEN	CECT ABDOMEN	CLINICAL DIAGNOSIS
77	present	absent	present	nil	tenderness	2800	90	6	2.5	1.5	0	42	36	28	35	6	14	air under diaphragm	No	ileal perforation
78	present	present	present	sluggish	normal	18000	86	10	3.1	0.9	0	162	188	212	187	-26	-50	air under diaphragm	No	colonic perforation
79	present	present	present	sluggish	normal	18000	82	13	2	2	1	360	400	320	360	-40	40	-	Appendiceal Perforation	appendicular perforation
80	present	present	present	nil	normal	20000	88	10	1.2	0.8	0	160	194	176	177	-34	-16	air under diaphragm	No	colonic perforation
81	present	present	present	sluggish	normal	16000	82	14	2.4	0.6	1	96	134	194	141	-38	-98	-	Appendiceal Perforation	Appendiceal Perforation
82	present	present	present	nil	tenderness	19500	88	6	4.2	0.8	1	156	174	189	173	-18	-33	air under diaphragm	No	peptic perforation
83	present	present	present	nil	normal	17000	89	8	2	1	0	170	195	223	196	-25	-53	air under diaphragm	No	Appendiceal Perforation
84	present	present	present	sluggish	normal	18,000	80	15	4	1	0	180	202	234	205	-22	-54	air under diaphragm	No	ileal perforation
85	present	present	present	nil	normal	22000	90	6	2.5	0.5	1	110	166	194	157	-56	-84	air under diaphragm	No	peptic perforation
86	present	present	present	sluggish	normal	17200	83	13	3.3	0.7	0	120.4	174	217	170	-53.6	-96.6	air under diaphragm	No	ileal perforation
87	present	present	present	sluggish	normal	16500	80	15	4	1	0	165	202	289	219	-37	-124	air under diaphragm	No	Appendiceal Perforation
88	present	present	present	sluggish	tenderness	15000	88	7	3	2	0	300	322	375	332	-22	-75	air under diaphragm	No	colonic perforation
89	present	present	present	sluggish	normal	16000	84	12	3	1	0	160	202	264	209	-42	-104	air under diaphragm	No	colonic perforation
90	present	present	present	nil	tenderness	19500	88	6	4.2	0.8	1	156	102	88	115	54	68	air under diaphragm	No	peptic perforation
91	present	present	present	sluggish	normal	14000	82	14	3	1	0	140	179	224	181	-39	-84	air under diaphragm	No	ileal perforation
92	present	present	present	nil	normal	15000	83	13	3	1	0	150	194	172	172	-44	-22	air under diaphragm	No	colonic perforation
93	present	absent	present	nil	normal	4600	84	10	4	1	1	46	38	36	40	8	10	air under diaphragm	No	peptic perforation
94	present	present	present	nil	normal	18,000	80	15	4.4	0.6	0	108	174	223	168	-66	-115	air under diaphragm	No	colonic perforation
95	present	present	present	sluggish	normal	17000	89	8	2	1	0	170	195	223	196	-25	-53	air under diaphragm	No	peptic perforation
96	present	present	present	nil	tenderness	16000	82	14	2.4	1.6	1	234	248	214	232	-14	20	air under diaphragm	No	peptic perforation
97	present	present	present	sluggish	normal	6000	87	10	2.1	0.9	0	54	66	41	54	-12	13	air under diaphragm	No	colonic perforation
98	present	present	present	sluggish	normal	14400	88	10	2.4	0.8	0	115	168	208	164	-53	-93	air under diaphragm	No	ileal perforation
99	present	present	present	nil	normal	16000	82	14	2.4	1	1	160	144	112	139	16	48	air under diaphragm	No	colonic perforation
100	present	present	present	nil	tenderness	22000	87	10	2.1	1.6	0	352	320	286	319	32	66	air under diaphragm	No	appendicular perforation
101	present	present	present	nil	normal	11000	86	9	3.8	1.2	0	132	189	242	188	-57	-110	-	appendicular perforation	appendicular perforation

S.No	SURGERY	DURATION OF SURGERY (HRS)	POST OPMorbidity 1. Wound infection	2. Intra abdominal collection	3. Pneumonia or lung atelectasis	4. Acute myocardial infarction or heart failure	5. Acute renal failure	6. Urinary tract infection	HOSPITAL STAY (DAYS)	Absolute eosinophilic count at the time of discharge	OVERALL MORTALITY	OVERALL MORBIDITY
77	perforation repair and proximal illeostomy	3	NO	NO	NO	NO	NO	NO	9	-	YES	NO
78	Exploratory laparotomy with proximal loop ileostomy	3	NO	NO	NO	NO	NO	NO	-	252	NO	NO
79	appendectomy	3	YES	NO	NO	NO	NO	NO	10	364	NO	YES
80	repair and proximal colostomy	5	YES	NO	YES	NO	YES	NO	12	164	NO	YES
81	appendectomy	2	NO	NO	NO	NO	NO	NO	7	243	NO	NO
82	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	NO	NO	NO	NO	NO	7	256	NO	NO
83	appendectomy	3.5	NO	NO	NO	NO	NO	NO	5	284	NO	NO
84	Resection anastomosis	2.5	NO	NO	NO	NO	NO	NO	7	289	NO	NO
85	Modiied Graham's Patch repair with feeding jejunostomy	3	NO	NO	NO	NO	NO	NO	6	234	NO	NO
86	Exploratort Laparotomy with primary repair of the perforation	3	NO	NO	NO	NO	NO	NO	6	264	NO	NO
87	appendectomy	3.5	NO	NO	NO	NO	NO	NO	6	312	NO	NO
88	Hartmann's Procedure	4	NO	NO	NO	NO	NO	NO	5	402	NO	NO
89	repair and proximal colostomy	4	NO	NO	NO	NO	NO	NO	6	294	NO	NO
90	Modiied Graham's Patch repair with feeding jejunostomy	3	YES	NO	NO	NO	YES	NO	11	82	NO	YES
91	Resection anastomosis	3	NO	NO	NO	NO	NO	NO	7	253	NO	NO
92	Hartmann's Procedure	3	NO	NO	YES	NO	YES	NO	8	176	NO	YES
93	Modified Graham's Patch Repair	2.5	NO	NO	NO	NO	NO	NO	12	-	YES	NO
94	Exploratory laparotomy with proximal loop ileostomy	3	NO	NO	NO	NO	NO	NO	6	279	NO	NO
95	Modified Graham's Patch Repair	3	NO	NO	NO	NO	NO	NO	7	296	NO	NO
96	Modified Graham's Patch Repair	4	YES	NO	NO	YES	NO	NO	8	154	NO	YES
97	Resection anastomosis	3.5	YES	NO	YES	YES	NO	NO	7	-	YES	YES
98	Resection anastomosis	3.5	NO	NO	NO	NO	NO	NO	5	243	NO	NO
99	Hartmann's Procedure	3	YES	NO	YES	NO	NO	NO	6	152	NO	YES
100	appendectomy	4	NO	NO	NO	NO	NO	YES	6	240	NO	YES
101	appendectomy	3	NO	NO	NO	NO	NO	NO	7	292	NO	NO

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No. BFUHS/2K21p-TH/ 127

Date 25/11/22

The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.10.2021 has approved the plan of thesis of **Aseem Gumber,** a student of your college for **MS General Surgery** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis

To evaluate the role of absolute eosinophil count as a prognostic marker in patients of perforation peritonitis.

Supervisor

Dr. Ravinder Pal Singh
Prof.,
Dept. of Surgery,
DMCH, Ludhiana.

Co-Supervisor

Dr. Atul Mishra
Prof.,
Dept. of Surgery,
DMCH, Ludhiana.

Dr. Amandeep S. Nar
Assoc. Prof.,
Dept. of Surgery,
DMCH, Ludhiana.

The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

S P Singh

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

121. If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.

122. The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.

TO EVALUATE THE ROLE OF ABSOLUTE EOSINOPHIL COUNT AS A PROGNOSTIC MARKER IN PATIENTS OF PERFORATION PERITONITS

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

Master of Surgery

In

GENERAL SURGERY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

March 2021

Dr. Aseem Gumber

DEPARTMENT OF GENERAL SURGERY
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA
LUDHIANA, PUNJAB-141001

ABSTRACT OF PLAN OF THESIS

Title	To Evaluate The Role Of Absolute Eosinophil Count As A Prognostic Marker In Patients Of Perforation Peritonitis
For the degree of	M.S. General Surgery
Name of the candidate	Dr Aseem Gumber
Supervisor	Dr. Ravinder Pal Singh, Professor, Department of Surgery, DMCH, Ludhiana
Co-supervisor	Dr. Atul Mishra, Professor, Department of Surgery, DMCH, Ludhiana Dr. Amandeep S. Nar, Associate Professor, Department of Surgery, DMCH, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Peritonitis is the commonest cause of sepsis in patients admitted in surgical wards in developing countries. Cost constraints prevent routine use of expansive tests like Procalcitonin assay in developing countries. Eosinopenia has been, recently, found to have strong association with inflammatory-syndrome-associated bacterial infectious diseases. This prompted us to prospectively assess the diagnostic value of eosinopenia as a prognostic marker in perforation peritonitis patients.

Aim of Study: To identify high risk patients and correlate mortality with eosinophilic count.

Materials & methods: This observational study will be conducted in Department of Surgery, Dayanand Medical College & Hospital, Ludhiana after approval by Institutional Ethics Committee on patients coming to casualty or OPD with perforative peritonitis from 01/02/2021 to 30/07/22. A written informed consent will be obtained from all patients or their legal representatives.

Clinical Significance: A rapid and persistent decrease in the numbers of circulating eosinophils is a distinctive aspect of physiological response to acute inflammation. Eosinopenia (<150cells/dl) may be the result of migration of eosinophils into the inflammatory site due to release of the chemotactic factors. Recent reports have shown that eosinopenia as a marker of sepsis. This promoted us to assess the diagnostic value of eosinopenia as mortality marker in patients with perforation peritonitis. This will allow us to timely identify the patients at risk and we will be able to associate disease outcome with absolute eosinophil count and correlate mortality with eosinophilic count.

Keywords: Absolute eosinophil count, Perforation peritonitis

(Signature)	(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"TO EVALUATE THE ROLE OF ABSOLUTE EOSINOPHIL COUNT AS A PROGNOSTIC MARKER IN PATIENTS OF PERFORATION PERITONITIS"** exist in the Department of Surgery, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate herself under our supervision and guidance.

Supervisor

Dr. Ravinder Pal Singh
Professor
Department of Surgery
DMCH, Ludhiana

Co-Supervisor

Dr. Atul Mishra
Professor
Department of Surgery
DMCH, Ludhiana

Dr. Amandeep S Nar
Associate Professor
Department of General Surgery
DMCH, Ludhiana

Head of the Department

Dr. Jaspal Singh
Professor & Head
Department of Surgery
DMCH, Ludhiana

Place : Ludhiana

Date : 30/3/21

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Aseem Gumber
Department	General Surgery
Topic of Thesis	TO EVALUATE THE ROLE OF ABSOLUTE EOSINOPHIL COUNT AS A PROGNOSTIC MARKER IN PATIENTS OF PERFORATION PERITONITIS
Likely date of appearing in PG examination	MAY 2023
Date of enrollment	26th July, 2020
Name of Head of Department	Dr. Jaspal Singh
1) Supervisor **2) Co-Supervisor**	Dr. Ravinder Pal Singh Dr. Atul Mishra Dr.Amandeep Singh Nar
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)	Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)
Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal & Professor & Head, Cardiology)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / No

Dr. Sandeep Puri
Chairperson
Research Committee

Approved: Yes / ~~No~~

Prof. Arvind Malhotra
Chairman
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	ASEEM GUMBER H.NO.6732, NEAR BARADARI, GURU TEG BAHADUR NAGAR, NAKODAR, JALANDHAR, PUNJAB
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL,LUDHIANA
3	COURSE OF STUDY AND SUBJECT	M.S. GENERAL SURGERY
4	DATE OF ADMISSION OF COURSE	26 JULY 2020
5	TITLE OF THE TOPIC	TO EVALUATE THE ROLE OF ABSOLUTE EOSINOPHIL COUNT AS A PROGNOSTIC MARKER IN THE PATIENTS OF PERFORATION PERITONITIS

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Perforation peritonitis is the commonest surgical emergency in India. In spite of advances in diagnosis, intensive care treatment, surgical techniques and antimicrobial therapy, the management of perforation peritonitis continues to be challenging for the surgeons.

Peritonitis is the commonest cause of sepsis in developing countries. The etiological spectrum of perforation peritonitis in India differs significantly from its western counterparts. It is commonly seen in younger age groups. The site of perforation in Indian patient population most commonly involves the proximal part of gastrointestinal tract whereas it is distal in the western countries. Etiological factors also show a wide geographical variation.

In India the common causes of perforation are peptic ulcer, typhoid followed by appendicular and tubercular perforations.

The most important factors responsible for the mortality from perforative peritonitis are septicaemia and shock. Unfortunately, despite undertaking appropriate treatment measures, mortality rates are very high ranging up to 40%.

In developing countries like India, under-developed infrastructure, inadequate diagnostic modalities and cost- restraints prevent the routine use of expensive tests like procalcitonin assay. Consequently, patients present to the tertiary hospitals at an advanced critical stage, thus significantly increasing the morbidity and mortality rates of the disease.

This urged us to look for an affordable, accurate and accessible tool which could aid in predicting the severity of the disease and its survival outcome thus helping in reducing the morbidity and mortality in patients with perforative peritonitis.

A rapid and persistent decrease in the numbers of circulating eosinophils is a distinctive aspect of physiological response to acute inflammation. Eosinophils are considered as the homeostatic regulators of inflammation. They leave the circulating blood when

adrenal cortical hormone increases in situations of stress and insult. Functionally this means that the eosinophils attempt to suppress inflammatory tissue reaction in order to prevent the excessive spread of inflammation. Early diagnosis with prompt surgical intervention and aggressive preoperative and postoperative management is essential to reduce the morbidity and mortality from multiorgan system failure resulting from untreated peritonitis.

Because eosinophil count is routinely done as a part of the complete blood count, it does not entail any extra effort or expenditure. Hence, it not only has the necessary sensitivity and specificity but also has the "ease" and "cost effectiveness" not seen with other markers for inflammation. The precocity and precision with which the eosinophil trend follows the stages of the inflammation, prompted us to study in depth the value of the assay of these cells as a reliable prognostic marker for monitoring perforative peritonitis. The concept is clinically very appealing and warrants larger prospective

6.2 REVIEW OF LITERATURE

PATHOPHYSIOLOGY

Peritonitis is defined as inflammation of the peritoneum & peritoneal cavity. The peritoneum, which is an otherwise sterile environment, reacts to a variety of pathologic stimuli with a fairly uniform inflammatory response.[1] Depending on the underlying pathology, the resultant peritonitis may be infectious or sterile (i.e. chemical).

Peritonitis is most often caused by introduction of an infection into the otherwise sterile peritoneal environment through organ perforation, but it may also result from other irritants, such as foreign bodies, bile from a perforated gall bladder or a lacerated liver, or gastric acid from a perforated ulcer. Women also experience localized peritonitis from an infected fallopian tube or a ruptured ovarian cyst.

PERFORATION PERITONITIS

Perforation peritonitis is by far the most common form of peritonitis encountered in clinical practice. It is caused by perforation of a hollow visceral organ with bacterial inoculation of the peritoneal cavity. The amount of spillage, however, does not influence development of peritonitis.

The pathogens involved differ in the proximal and distal GI tract perforation. Gram-positive organisms predominate in the upper GI tract, with a shift toward gram-negative organisms in the upper GI tract in patients on long-term gastric acid suppressive therapy.

Contamination from a distal small bowel or colon source initially may result in the release of several hundred bacterial species (and fungi); host defences quickly eliminate most of these organisms. The resulting peritonitis is almost always polymicrobial, containing a mixture of aerobic and anaerobic bacteria with a predominance of gram-negative organisms.

The clinicians need sufficient tests to promptly diagnose sepsis as early diagnosis and treatment will significantly reduce the mortality and morbidity. An early diagnosis of septicemia without microbial culture would ensure early action to reduce the patient mortality.

It is well known fact that in acute infection/ inflammation there is rapid decrease in the number of circulating eosinophils[2-4]

"This marked reduction in the number of circulating eosinophil leucocytes in acute infection was first described by Zappert in 1893"[5]and widely used in the last century as a useful diagnostic sign . This was followed later by the information that eosinopenia is part of the normal response to stress, it was said that eosinopenia in acute infection is a secondary response to stress induced by the infection[6-7]The value of this old marker of acute infection was tested by Gill and colleagues". [8]

A study by Swaminathan SP et al published in International Journal of Surgery was done in Madras Medical College, Chennai [9]

A total of 104 patients from October 2017 to October 2018 were chosen and were allocated into two groups based on the outcome as mortality or survival. 88 patients were in the survival group and 16 patients were in the mortality group. The Absolute eosinophil count distribution in both these groups were recorded.

From the result it was concluded that AEC is a reliable marker of survival and it allows timely identification of high-risk patients. It can be used as a marker for risk stratification in perforative peritonitis patients. AEC has the necessary sensitivity and specificity in addition to easy methodology and cost effectiveness as seen with other markers of sepsis

Gold standard test for the identification of sepsis has not been identified so far but Procalcitonin is found to play promising role in septicemia and it is found to complement clinical findings and lab test results in cases of septicemia.[10-12] The disadvantage of procalcitonin assay is its cost, and because of the high cost this test does not gain popularity in developing countries and it cannot done in our hospitals.

There is one study conducted by Jagdeesh et al using eosinopenia as a marker for survival in patients with peritonitis[13] They have used ROC/ AUROC to compare the diagnostic accuracy of JPS,CRP and Absolute eosinophil count(AEC).They concluded that AEC allows

timely identification of patients at high risk for sepsis related mortality jn patients with peritonitis.

Smithson *et al.*[14] showed no correlation between eosinopenia and infections. Setterberg *et al.*[15] also indicated that eosinopenia is not a valuable marker for infection. But resonating with our study, the research published by Abidi et al also proposed eosinopenia as an early marker of mortality in critically ill patient. [16-17]
The research further stated eosinopenia to be better marker of blood stream infections in critically ill patients than the commonly used CRP and procalcitonin. [18] A few scientists have even evaluated the usefulness of AEC in differentiating between sepsis and SIRS [12]. The role of AEC has been contradicted by other authors through their published literature, where they found CRP and procalcitonin assays better markers than eosinopenia [19]. On the other hand, Kendall et al. [20] reported albumin to be an independent predictor of mortality in ICU patients with sepsis. Similar findings were also suggested by Takegawa et al. who in addition to serum albumin also suggested the role of total protein levels in predicting mortality of critically ill patients. [21] Karakonstantis et al [22] and Hwang et al [23] conducted a research on the neutrophil-to-lymphocyte count ratio as a potential prognostic marker just like AEC.
However, while reviewing the published research articles, we found that many questioned the capacity of AEC to discriminate infection from controls, [24, 25] they attributed it to the underlining factors like geographical variation. Since each surgical/medical unit serves a different patient population, the authors suggested that each prognostic marker must be validated as a result. Moreover, it may even have different set of cut-off values to ensure that the model is apt and accurate for the patient material involved, before it is accepted as quality standard. Undoubtedly, the septic syndrome and the body's inflammatory response are far too heterogeneous and variable to be limited to a single absolute cutoff of any surrogate marker.

Different microbes might stimulate diverse responses, resulting in a distinct up/downregulation of circulating biomarkers and mediators [26] Eosinophil count is routinely done in patients, rather more often in critically ill patients as part of complete blood count, it holds the advantage of being economical with no extra efforts. Hence, AEC not only has the sensitivity and specificity but also has the "ease" and "cost effectiveness" not seen with other markers for sepsis

6.3 AIMS AND OBJECTIVES OF THE STUDY

To timely identify high risk patients so as to correlate morbidity and mortality with absolute eosinophilic count

7. MATERIALS AND METHODS

7.1 Source of Data

Data shall be collected from all patients with Perforation peritonitis, who are admitted during the given time period (1st March 2021 to 31st May 2022) will be taken for study considering the inclusion and exclusion criteria.

7.2 METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA-

- Patients with secondary bacterial peritonitis due to hollow viscous perforation.

EXCLUSION CRITERIA-

1. Spontaneous bacterial peritonitis
2. Malignant perforation.
3. Traumatic perforation
4. Non-resusticable patients
5. Post surgical leak.

METHOD OF COLLECTION OF DATA:

All the patients coming for acute abdomen in OPD or casualty with clinical signs of peritonitis will be investigated for perforation by having chest x-ray and x ray erect abdomen. After confirmation of perforation peritonitis, requisition for eosinophilic and absolute eosinophilic count will be sent along other routine investigations.

At the time of admission, 5 mL of venous blood will be collected in EDTA tube for the determination of Absolute Eosinophil Count (AEC)

The absolute eosinophil count is determined in the Neubaeur counting chamber by counting the number of eosinophils per 100 white blood cells by using It is then multiplied by the white blood cell count of the patient.

Sample collection duration: At the time of onset of symptoms
24 hours
72 hours
At the time of discharge

The reports of these along with other vital parameters –Pulse, BP, Temperature will be recorded everyday.

The correlation between eosinophilic count and the final outcome that is discharge or death will be studied for any correlation.

Duration of the study: 15 months from 1st March 2021 to 31st May 2022

OUTCOME

To conclude that AEC is a reliable marker of survival and it allows timely identification of high risk patients and it can be used as a marker for risk stratification in perforation peritonitis patients.

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. Comparison of quantitative variables between the study groups will be done using Student t-test and Mann Whitney U test for independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi square ($\chi 2$) test will be performed and exact test was used when the expected frequency is less than 5. A probability value (p value)less than 0.05 will be considered statistically significant.All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

The study does not require any investigation or intervention other than those routinely done for every surgery.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith

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PROFORMA

Sr. No:

NAME :	
AGE /SEX:	
ADDRESS WITH CONTACT NUMBER:	
MRD NO:	
DATE OF ADMISSION:	DATE OF SURGERY:

HISTORY

HISTORY OF PRESENTING ILLNESS

PAIN	**FEVER**
Origin:	Temperature:
Duration:	Duration :
Location:	Pattern:
Character:	

Signature of Candidate **Signature of Supervisor**

Any Other Relevant History	NAUSEA/ VOMITING Onset: Duration:

PAST HISTORY

Previous Surgery:

Previous Hospitalization:

Whether A Known Case Of DM/ TB/ Hypertension/
Asthma/ Epilepsy/ Cardiac Illness :

Signature of Candidate

Signature of Supervisor

CLINICAL EXAMINATION

GENERAL EXAMINATION	
APPEARANCE: VITALS: PR: BP: RR: Urine Output:	SENSORIUM:
SYSTEMIC EXAMINATION	
CVS:	RS:
PER ABDOMEN:	
Tenderness :	Guarding:
Rigidity:	Bowel sounds :

Signature of Candidate

Signature of Supervisor

INVESTIGATIONS:

HEMATOLOGICAL				
Date/ SNo.	TLC	DLC	AEC	PTI/INR

RFT ABG	LFT

Signature of Candidate **Signature of Supervisor**

RADIOLOGICAL INVESTIGATIONS
CXR: XRAY ABDOMEN: CECT ABDOMEN:

CLINICAL DIAGNOSIS:
SURGERY:

Signature of Candidate

Signature of Supervisor

POST OPERATIVE	
Wound infection Any 1 of the following: 1.Purulent wound discharge 2.Erythema >1cm from wound edges 3.Localised swelling pain or heat 4.Patient reporting increased tenderness Occurring within 30 days of Surgery	Intra- abdominal collection 1.Fever 2.TLC counts 3.Sugggestive CT findings
Pneumonia or lung atelectasis: 1.Xray changes 2..Respiratory rate 3.Blood urea nitrogen 4.CT changes	Acute myocardial infarction or heart failure: 1.ECG changes 2.Pain in chest 3.Troponon T levels.
Acute renal failure 1.Urine output 2.Creatinine 3.GFR	Urinary tract infection: 1.Fever (≥38°C) or chills. 2.New or increased burning pain on urination, frequency, or urgency. 3.New flank pain or suprapubic pain or tenderness
HOSPITAL STAY:	
AEC at the time of discharge:	
OVERALL MORBIDITY AND MORTALITY:	

Signature of Candidate **Signature of Supervisor**

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o..... am willing to participate in the study **"TO EVALUATE THE ROLE OF ABSOLUTE EOSINOPHIL COUNT AS A PROGNOSTIC MARKER IN PATIENTS OF PERFORATION PERITONITIS"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

....

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"TO EVALUATE THE ROLE OF ABSOLUTE EOSINOPHIL COUNT AS A PROGNOSTIC MARKER IN PATIENTS OF PERFORATION PERITONITIS"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"TO EVALUATE THE ROLE OF ABSOLUTE EOSINOPHIL COUNT AS A PROGNOSTIC MARKER IN PATIENTS OF PERFORATION PERITONITIS" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ